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=> fil reg
FILE 'REGISTRY' ENTERED AT 10:48:11 ON 27 AUG 2003
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STRUCTURE FILE UPDATES:   25 AUG 2003  HIGHEST RN 573649-48-6
DICTIONARY FILE UPDATES:  25 AUG 2003  HIGHEST RN 573649-48-6

TSCA INFORMATION NOW CURRENT THROUGH JANUARY 6, 2003

  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.

Crossover limits have been increased.  See HELP CROSSOVER for details.

Experimental and calculated property data are now available.  See HELP
PROPERTIES for more information.  See STNote 27, Searching Properties
in the CAS Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

=> d sta que l20
L12             STR
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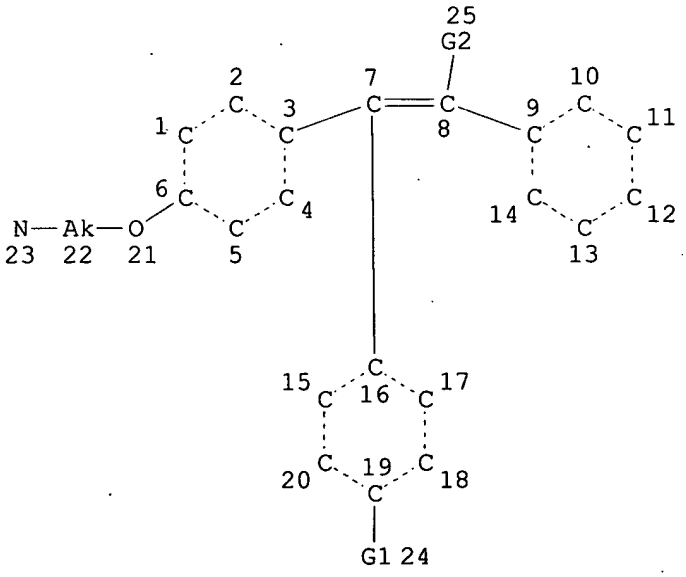

Jan Delaval
Reference Librarian
Biotechnology & Chemical Library
CM1 1E07 – 703-308-4498
jan.delaval@uspto.gov

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VAR G1=H/OH
VAR G2=H/AK
NODE ATTRIBUTES:
NSPEC   IS RC     AT  23
CONNECT IS M1  RC AT  10
CONNECT IS M1  RC AT  11
CONNECT IS M1  RC AT  12
CONNECT IS M1  RC AT  13
CONNECT IS M1  RC AT  14
CONNECT IS M1  RC AT  23
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
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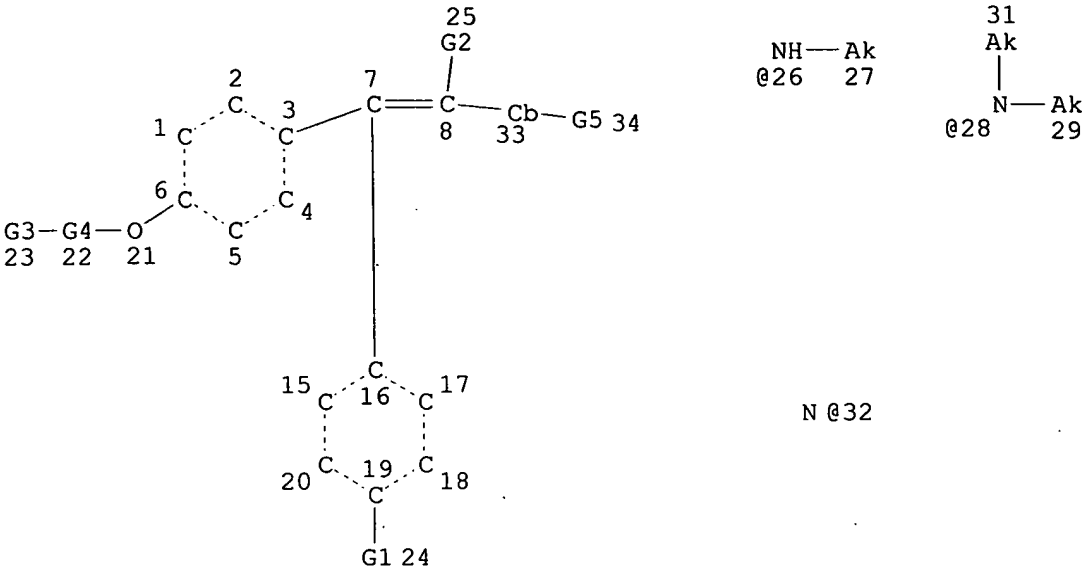
NUMBER OF NODES IS 25

STEREO ATTRIBUTES: NONE
L14 SCR 2039 OR 2043 OR 2054
L16 422 SEA FILE=REGISTRY CSS FUL L12 NOT L14
L17 STR



VAR G1=H/OH
VAR G2=H/AK
VAR G3=26/28/32
REP G4=(2-2) CH2
VAR G5=H/X/OH/AK
NODE ATTRIBUTES:
NSPEC IS R AT 32
CONNECT IS M1 RC AT 32
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 27

STEREO ATTRIBUTES: NONE
L20 235 SEA FILE=REGISTRY SUB=L16 CSS FUL L17

100.0% PROCESSED 421 ITERATIONS 235 ANSWERS
SEARCH TIME: 00.00.01

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SET COST OFF

FILE 'HCAPLUS' ENTERED AT 09:48:08 ON 27 AUG 2003
L1 2 S US20020065325/PN OR (WO2002-US26120# OR WO2000-US4892# OR US2
SEL RN

FILE 'REGISTRY' ENTERED AT 09:50:12 ON 27 AUG 2003
L2 17 S E1-E17
L3 1 S L2 AND C26H29NO

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L4          109 S C26H29NO/MF AND 46.150.18/RID AND 3/NR
L5           24 S L4 AND BUTEN? AND PHENOXY AND DIMETHYL AND ETHANAMINE
L6            5 S L5 NOT (LABELED OR (D OR T)/ELS OR 14C#)
L7            4 S L6 NOT 2 BUTENYL
L8            4 S L3,L7
                SEL RN
L9           24 S E18-E21/CRN
L10          14 S L9 NOT (COMPD OR WITH OR MXS/CI)
L11          18 S L8,L10
                STR
L12
L13          18 S L12 CSS SAM
                SCR 2039 OR 2043 OR 2054
L14
L15          16 S L12 NOT L14 CSS
L16         422 S L12 NOT L14 CSS FUL
                SAV L16 JKIM930/A
L17             STR L12
L18          11 S L17 CSS SAM SUB=L16
L19           4 S L16 AND C6-C6/ES
L20         235 S L17 CSS FUL SUB=L16
                SAV L20 JKIM930A/A
L21         182 S L20 AND 3/NR
L22          35 S L20 AND 4/NR
L23          18 S L20 NOT L11,L21,L22
L24           9 S L23 NOT (NC5/ES OR CLO4 OR MXS/CI OR C60H66N4O4 OR C20H8BR4O5

     FILE 'HCAPLUS' ENTERED AT 10:14:03 ON 27 AUG 2003
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L26        7288 S TAMOXIFEN# OR ICI47699 OR ICI()(47699 OR 47 699)
L27        5795 S L19 OR L21 OR L22 OR L24
L28        7816 S L25-L27
                E BLOOD VESSEL/CT
L29       65303 S E3-E59
L30       13171 S E60-E96
L31        6190 S E109-E115
                E E3+ALL
L32      143069 S E5,E4+NT
                E E25+ALL
L33       92479 S E4,E5,E3+NT
                E E114+ALL
                E E28+ALL
L34        4260 S E3
                E E8+ALL
                E E30+ALL
L35        5383 S E3
                E E10+ALL
L36        9115 S E4
                E IMPOTENCE/CT
                E E3+ALL
L37        1385 S E2
                E ERECT/CT
                E E10+ALL
                E PENI/CT
                E PENIL/CT
                E E7+ALL
L38         446 S E2
                E PENIS/CT
L39        1507 S E3-E8
                E E3+ALL
L40        1902 S E6+NT
L41         324 S L28 AND L29-L40
L42         177 S L41 AND (PD<=19990226 OR PRD<=19990226 OR AD<=19990226)
L43         121 S L42 AND L25
L44         125 S L42 AND L27
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L45          125 S L43,L44
L46           52 S L42 NOT L45
L47            2 S L46 AND (NOREPINEPHRIN? OR CARDIOVASCULAR PATHOLOG?)/TI
                 E LAMB F/AU
L48            7 S E3,E11
L49           22 S E29,E31
                 E SCHUTTE B/AU
L50           57 S E3,E4,E7-E9
                 E YANG B/AU
L51          787 S E3-E24
                 E YANG BAO/AU
L52           18 S E3
L53           21 S E60
L54            4 S L28 AND L48-L53
L55           92 S L45 AND (PHARMACOL? OR PHARMACEUT?)/SC,SX
L56         2386 S (L11 OR L19 OR L21 OR L22 OR L24)(L)THU/RL
L57          551 S (L11 OR L19 OR L21 OR L22 OR L24)(L)(DMA OR PAC OR PKT)/RL
L58         2620 S (L11 OR L19 OR L21 OR L22 OR L24)(L)(BAC OR BCP OR BPR OR BSU
L59          104 S L45 AND L56-L58
L60          119 S L55,L59
L61            6 S L45 NOT L60
                 SEL L60 DN AN 3 8 9 27 74 81 93 119
L62            8 S L60 AND E1-E24
L63           11 S L1,L47,L54,L62 AND L25-L62
L64            4 S L63 AND (CL OR CL3 OR CLC3)(L)CHANNEL?
L65            3 S L63 AND (CHANNEL OR ION OR CHLORIDE OR CHLORIN?)(L)BLOCK?
L66            4 S L64,L65
L67            4 S L63 AND CHLORIDE(L)CHANNEL
L68            4 S L66,L67

     FILE 'REGISTRY' ENTERED AT 10:43:58 ON 27 AUG 2003
L69            1 S NOREPINEPHRINE/CN
L70            5 S C8H11NO3/MF AND NOREPINEPHRIN?
L71            3 S L70 NOT LABELED
L72            3 S L69,L71

     FILE 'HCAPLUS' ENTERED AT 10:45:21 ON 27 AUG 2003
L73           27 S L72 AND L28
L74           19 S NOREPINEPHRIN? AND L28
L75           10 S L73,L74 AND L42
                 SEL DN AN 3 5 8 9 10
L76            5 S L75 NOT E25-E39
L77            6 S L68,L76
L78            6 S L63 NOT L77
L79           12 S L77,L78 AND L1,L25-L68,L73-L78

     FILE 'REGISTRY' ENTERED AT 10:48:11 ON 27 AUG 2003

=> fil hcaplus
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FILE COVERS 1907 - 27 Aug 2003 VOL 139 ISS 9
FILE LAST UPDATED: 25 Aug 2003 (20030825/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> d l79 all hitstr tot

L79 ANSWER 1 OF 12 HCAPLUS COPYRIGHT 2003 ACS on STN
AN **2002:409273** HCAPLUS
DN **137:722**
TI Use of **CLC3 chloride channel blockers** to modulate vascular tone
IN **Lamb, Fred S.; Schutte, Brian C.; Yang, Baoli**
PA USA
SO U.S. Pat. Appl. Publ., 46 pp., Cont.-in-part of U. S. Ser. No. 512,926.
CODEN: USXXCO
DT Patent
LA English
IC ICM A61K031-137
ICS A61K031-55; A61K031-445; A61K031-40
NCL 514651000
CC 1-8 (**Pharmacology**)
Section cross-reference(s): **63**
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2002065325	A1	20020530	US 2001-930105	20010815 <--
WO 2003015614	A2	20030227	WO 2002-US26120	20020815 <--

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 1999-121727P	P	19990226	<--
US 2000-512926	A2	20000225	<--
US 2001-930105	A	20010815	

OS MARPAT 137:722
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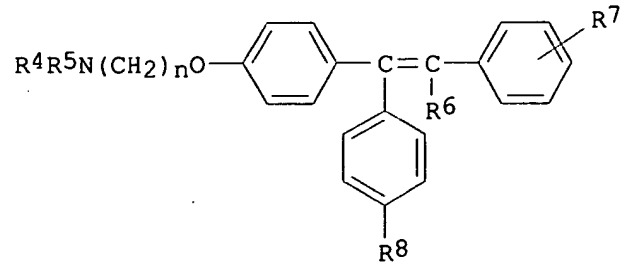


I

AB The invention discloses the use of **chloride channel**

blocking compd. I (R4= H, lower alkyl radical; R5= lower alkyl radical; or R4 and R5 connected with adjacent nitrogen to form a heterocyclic radical; R6= H, lower alkyl radical; R7=H, halogen, OH, lower alkyl radical, buta-1-3-dienyl radical which together with adjacent Ph forms a naphthyl radical; R8=H, OH; n=2) for the modulation of vascular tone in a patient having compromised vascular tissue. The present invention also provides methods for the modulation of vascular tone in a patient having compromised vascular tissue, with the administration of a **chloride channel blocking** agent or a pharmaceutically acceptable salt thereof.

ST **chloride channel CLC3 blocker**
blood vessel endothelium damage

IT **Chloride channel**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**CLC3**; use of **CLC3 chloride channel blockers** to modulate vascular tone)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Clcn3; use of **CLC3 chloride channel blockers** to modulate vascular tone)

IT Brain
(cerebral cortex; use of **CLC3 chloride channel blockers** to modulate vascular tone)

IT **Ion channel blockers**
(**chloride**; use of **CLC3 chloride channel blockers** to modulate vascular tone)

IT **Artery, disease**
(coronary; use of **CLC3 chloride channel blockers** to modulate vascular tone)

IT Nerve, disease
Nerve, disease
(death; use of **CLC3 chloride channel blockers** to modulate vascular tone)

IT **Blood vessel, disease**
(**endothelium, injury**; use of **CLC3 chloride channel blockers** to modulate vascular tone)

IT **Blood vessel**
(**endothelium**; use of **CLC3 chloride channel blockers** to modulate vascular tone)

IT Neuroglia, disease
(gliosis; use of **CLC3 chloride channel blockers** to modulate vascular tone)

IT Brain
(hippocampus; use of **CLC3 chloride channel blockers** to modulate vascular tone)

IT **Sexual behavior**
(**impotence**; use of **CLC3 chloride channel blockers** to modulate vascular tone)

IT Drug delivery systems
(injections, i.v.; use of **CLC3 chloride channel blockers** to modulate vascular tone)

IT Electric current
(ionic, biol.; use of **CLC3 chloride channel blockers** to modulate vascular tone)

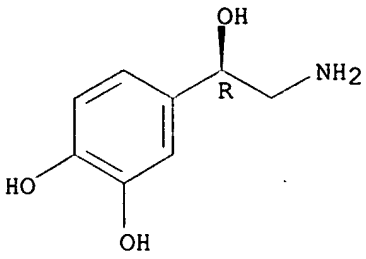
IT Cell death
Cell death
(neuron; use of **CLC3 chloride channel blockers** to modulate vascular tone)

IT Drug delivery systems
(oral; use of **CLC3 chloride channel blockers** to modulate vascular tone)

IT **Artery, disease**

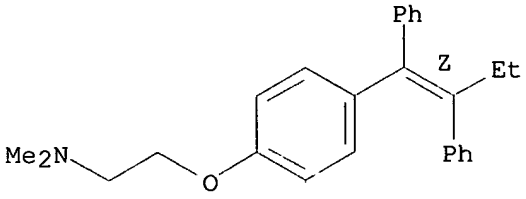
(restenosis; use of **CLC3 chloride channel blockers** to modulate vascular tone)
IT Eye
(retina; use of **CLC3 chloride channel blockers** to modulate vascular tone)
IT **Blood vessel**
(**smooth muscle**; use of **CLC3 chloride channel blockers** to modulate vascular tone)
IT Antidiabetic agents
Antihypertensives
Diabetes insipidus
Diabetes mellitus
Human
Hypertension
Mammalia
Mutation
Seizures
Surgery
Vasoconstriction
Vasodilators
(use of **CLC3 chloride channel blockers** to modulate vascular tone)
IT Estrogen receptors
Glial fibrillary acidic protein
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(use of **CLC3 chloride channel blockers** to modulate vascular tone)
IT **Penis**
(vascular, sympathetic tone; use of **CLC3 chloride channel blockers** to modulate vascular tone)
IT 50-99-7, D-Glucose, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(tolerance; use of **CLC3 chloride channel blockers** to modulate vascular tone)
IT **51-41-2, Norepinephrine** 141436-78-4, Protein kinase C
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(use of **CLC3 chloride channel blockers** to modulate vascular tone)
IT 2149-70-4, Nitro-L-Arginine 21829-25-4, Nifedipine
RL: PAC (Pharmacological activity); BIOL (Biological study)
(use of **CLC3 chloride channel blockers** to modulate vascular tone)
IT 61-68-7, Mefenamic acid 91-40-7 128-42-7, DNDS 530-78-9, Flufenamic acid 723-62-6, Anthracene-9-carboxylic acid 4394-00-7, Niflumic acid **10540-29-1, Tamoxifen** 51023-76-8, SITS 53005-05-3, DIDS 53108-00-2, IAA-94 107254-86-4, NPPB
RL: **PAC (Pharmacological activity)**; **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(use of **CLC3 chloride channel blockers** to modulate vascular tone)
IT **51-41-2, Norepinephrine**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(use of **CLC3 chloride channel blockers** to modulate vascular tone)
RN 51-41-2 HCAPLUS
CN 1,2-Benzenediol, 4-[(1R)-2-amino-1-hydroxyethyl]- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



IT **10540-29-1, Tamoxifen**
RL: **PAC (Pharmacological activity); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(use of **CLC3 chloride channel blockers** to modulate vascular tone)
RN 10540-29-1 HCAPLUS
CN Ethanamine, 2-[4-[(1Z)-1,2-diphenyl-1-butenyl]phenoxy]-N,N-dimethyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L79 ANSWER 2 OF 12 HCAPLUS COPYRIGHT 2003 ACS on STN
AN **2001:161506** HCAPLUS
DN **134:202697**
TI Prevention and treatment of cardiovascular pathologies with **tamoxifen** analogues
IN Grainger, David J.; Metcalfe, James C.; Kunz, Lawrence L.; Schroff, Robert W.
PA NeoRx Corporation, USA
SO U.S., 48 pp., Cont.-in-part of U.S. Ser. No. 478,936, abandoned.
CODEN: USXXAM
DT Patent
LA English
IC ICM A61K031-445
ICS A61K031-40; A61K031-38; A61K031-135
NCL 514319000
CC 1-8 (**Pharmacology**)
FAN.CNT 14

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 6197789	B1	20010306	US 1997-973570	19971205 <--
	US 5595722	A	19970121	US 1995-476735	19950607 <--
	US 5770609	A	19980623	US 1995-486334	19950607 <--
	US 6395494	B1	20020528	US 1995-477393	19950607 <--
	WO 9640098	A2	19961219	WO 1996-US10211	19960607 <--
	WO 9640098	A3	19970619		

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RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR,

IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA
US 2002068731 A1 20020606 US 2001-754775 20010104 <--
PRAI US 1995-476735 A2 19950607 <--
US 1995-477393 A2 19950607 <--
US 1995-478936 B2 19950607 <--
US 1995-486334 A2 19950607 <--
WO 1996-US10211 W 19960607 <--
US 1993-11669 B2 19930128 <--
US 1993-61714 B2 19930513 <--
US 1993-62451 B2 19930513 <--
US 1994-241844 A2 19940512 <--
US 1994-242161 A2 19940512 <--
US 1997-973570 A1 19971205 <--
OS MARPAT 134:202697
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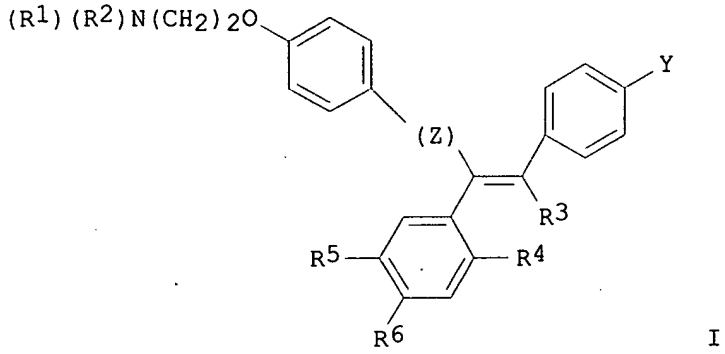

I

AB A method for treating or preventing cardiovascular pathologies by administering a compd. of the formula (I): wherein Z is C:O or a covalent bond; Y is H or O(C1-C4)alkyl, R1 and R2 are individually (C1 -C4)alkyl or together with N are a satd. heterocyclic group, R3 is Et or chloroethyl, R4 is H, R5 is I, O(C1 -C4)alkyl or H and R6 is I, O(C1 -C4)alkyl or H with the proviso that when R4, R5, and R6 are H, R3 is not ethyl; or a pharmaceutically acceptable salt thereof, effective to elevate the level of TGF-beta to treat and/or prevent conditions such as atherosclerosis, thrombosis, myocardial infarction, and stroke is provided. Useful compds. include idoxifene, toremifene or salts thereof. Further provided is a method for identifying an agent that elevates the level of TGF-beta. Another embodiment of the invention is an assay or kit to det. TGF-beta in vitro. Also provided is a therapeutic method comprising inhibiting smooth muscle cell proliferation assocd. with procedural vascular trauma employing the administration of **tamoxifen** or structural analogs thereof, including compds. of formula (I).

ST **tamoxifen** analog cardiovascular pathol TGF beta; smooth muscle proliferation inhibition **tamoxifen** analog; thrombosis inhibition **tamoxifen** analog TGF beta; myocardial infarction treatment **tamoxifen** analog TGF; stroke treatment **tamoxifen** analog TGF beta

IT Antiarteriosclerotics
(antiatherosclerotics; prevention and treatment of cardiovascular pathol. with **tamoxifen** analogs and inhibition of vascular smooth muscle proliferation in relation to elevation of TGF beta)

IT Medical goods
(catheters, drug delivery with; prevention and treatment of cardiovascular pathol. with **tamoxifen** analogs and inhibition

of vascular smooth muscle proliferation in relation to elevation of TGF beta)

IT **Artery**
(coronary, angioplasty, treatment of; prevention and treatment of cardiovascular pathol. with **tamoxifen** analogs and inhibition of vascular smooth muscle proliferation in relation to elevation of TGF beta)

IT Cardiovascular system
(disease; prevention and treatment of cardiovascular pathol. with **tamoxifen** analogs and inhibition of vascular smooth muscle proliferation in relation to elevation of TGF beta)

IT Lipids, biological studies
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(metabolic disorders, treatment of cardiovascular pathol. in; prevention and treatment of cardiovascular pathol. with **tamoxifen** analogs and inhibition of vascular smooth muscle proliferation in relation to elevation of TGF beta)

IT Cardiovascular agents
Drug delivery systems
(prevention and treatment of cardiovascular pathol. with **tamoxifen** analogs and inhibition of vascular smooth muscle proliferation in relation to elevation of TGF beta)

IT Proliferation inhibition
(proliferation inhibitors; prevention and treatment of cardiovascular pathol. with **tamoxifen** analogs and inhibition of vascular smooth muscle proliferation in relation to elevation of TGF beta)

IT **Artery, disease**
(restenosis, inhibition of; prevention and treatment of cardiovascular pathol. with **tamoxifen** analogs and inhibition of vascular smooth muscle proliferation in relation to elevation of TGF beta)

IT **Blood vessel**
(**smooth muscle**; prevention and treatment of cardiovascular pathol. with **tamoxifen** analogs and inhibition of vascular **smooth muscle** proliferation in relation to elevation of TGF beta)

IT **Artery, disease**
(**stenosis**, inhibition of; prevention and treatment of cardiovascular pathol. with **tamoxifen** analogs and inhibition of vascular smooth muscle proliferation in relation to elevation of TGF beta)

IT Medical goods
(stents, inhibition of restenosis from; prevention and treatment of cardiovascular pathol. with **tamoxifen** analogs and inhibition of vascular smooth muscle proliferation in relation to elevation of TGF beta)

IT Diabetes mellitus
(treatment of cardiovascular pathol. in; prevention and treatment of cardiovascular pathol. with **tamoxifen** analogs and inhibition of vascular smooth muscle proliferation in relation to elevation of TGF beta)

IT **Artery, disease**
(treatment of; prevention and treatment of cardiovascular pathol. with **tamoxifen** analogs and inhibition of vascular smooth muscle proliferation in relation to elevation of TGF beta)

IT Transforming growth factors
RL: ANT (Analyte); BPR (Biological process); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study); PROC (Process)
(.beta.-; prevention and treatment of cardiovascular pathol. with **tamoxifen** analogs and inhibition of vascular smooth muscle proliferation in relation to elevation of TGF beta)

IT **10540-29-1D, Tamoxifen**, analogs 82413-20-5, Droloxifene 84449-90-1, Raloxifene 89778-26-7, Toremifene

116057-66-0 116057-75-1, Idoxifene 116057-76-2
RL: **BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(prevention and treatment of cardiovascular pathol. with **tamoxifen** analogs and inhibition of vascular smooth muscle proliferation in relation to elevation of TGF beta)

RE.CNT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Allen; US 2914563 1959 HCAPLUS
(2) Anon; EP 0054168 1982 HCAPLUS
(3) Anon; EP 054168 A1 1982 HCAPLUS
(4) Anon; DE 4401554 1994 HCAPLUS
(5) Anon; DE 4320896 1995 HCAPLUS
(6) Anon; DE 4320898 1995 HCAPLUS
(7) Anon; Drug & Market Development 1994, V5, P121
(8) Anon; ICI Pharma 1992, P64033
(9) Anon; The Breast Cancer Letter 1994, V20, P4
(10) Crawley; US 4307111 1981 HCAPLUS
(11) Dewald; US 3288806 1966 HCAPLUS
(12) Elpern; US 3010965 1961
(13) Fildes; US 4235988 1980 HCAPLUS
(14) Harita; US 3940422 1976 HCAPLUS
(15) Harita; US 4070484 1978
(16) Harita; US Re32944 1989
(17) Jones; US 4133814 1979 HCAPLUS
(18) Jones; US 4418068 1983 HCAPLUS
(19) Magarian; US 4442119 1984 HCAPLUS
(20) Palopoli; US 3168565 1965 HCAPLUS
(21) Palopoli; US 3634517 1972 HCAPLUS
(22) Peters; US 4380635 1983 HCAPLUS
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(24) Spears; US 4512762 1985
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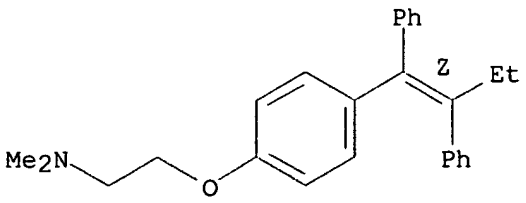
IT **10540-29-1D, Tamoxifen**, analogs
RL: **BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(prevention and treatment of cardiovascular pathol. with **tamoxifen** analogs and inhibition of vascular smooth muscle proliferation in relation to elevation of TGF beta)

RN 10540-29-1 HCAPLUS
CN Ethanamine, 2-[4-[(1Z)-1,2-diphenyl-1-butenyl]phenoxy]-N,N-dimethyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L79 ANSWER 3 OF 12 HCAPLUS COPYRIGHT 2003 ACS on STN
AN **2000:608566** HCAPLUS

DN **133:172188**
TI Methods to reduce the sensitivity of endothelially-compromised vascular smooth muscle
IN **Lamb, Fred S.**
PA University of Iowa Research Foundation, USA
SO PCT Int. Appl., 34 pp.
CODEN: PIXXD2
DT Patent
LA English
IC A61K031-00
CC 1-8 (**Pharmacology**)
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000050023	A2	20000831	WO 2000-US4892	20000226 <--

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRAI US 1999-121727P P 19990226 <--
OS MARPAT 133:172188
AB The present invention discloses materials and methods useful to treat sensitivity of endothelially-compromised vascular smooth muscle. In one embodiment, CLC3 blockers, particularly compds. of formula I are used to treat sensitivity.
ST vascular smooth muscle endothelial damage treatment **tamoxifen**; CLC3 blocker vascular smooth muscle endothelial damage
IT **Chloride channel**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(CLC-3; methods to reduce sensitivity of endothelially-compromised vascular smooth muscle using **CLC3 chloride channel blockers** such as **tamoxifen** in relation to use of other agents)
IT **Ion channel blockers**
(**chloride**, CLC-3; methods to reduce sensitivity of endothelially-compromised vascular smooth muscle using **CLC3 chloride channel blockers** such as **tamoxifen** in relation to use of other agents)
IT **Artery, disease**
(coronary, restenosis, agents for treatment of; methods to reduce sensitivity of endothelially-compromised vascular smooth muscle using **CLC3 chloride channel blockers** such as **tamoxifen** in relation to use of other agents)
IT **Artery, disease**
(coronary, vascular endothelial damage in, treatment of; methods to reduce sensitivity of endothelially-compromised vascular smooth muscle using **CLC3 chloride channel blockers** such as **tamoxifen** in relation to use of other agents)
IT **Blood vessel, disease**
(**diabetic angiopathy**, treatment of; methods to reduce sensitivity of endothelially-compromised vascular **smooth muscle** using **CLC3 chloride channel blockers** such as **tamoxifen** in relation to use of other agents)
IT **Blood vessel, disease**
(**endothelium, injury**; methods to reduce sensitivity

of endothelially-compromised vascular **smooth muscle** using **CLC3 chloride channel blockers** such as **tamoxifen** in relation to use of other agents)

IT Antidiabetic agents
Antihypertensives
Drug interactions
Vasodilators
(methods to reduce sensitivity of endothelially-compromised vascular smooth muscle using **CLC3 chloride channel blockers** such as **tamoxifen** in relation to use of other agents)

IT **Blood vessel**
(**smooth muscle**; methods to reduce sensitivity of endothelially-compromised vascular **smooth muscle** using **CLC3 chloride channel blockers** such as **tamoxifen** in relation to use of other agents)

IT Hypertension
Surgery
(vascular endothelial damage from, treatment of; methods to reduce sensitivity of endothelially-compromised vascular smooth muscle using **CLC3 chloride channel blockers** such as **tamoxifen** in relation to use of other agents)

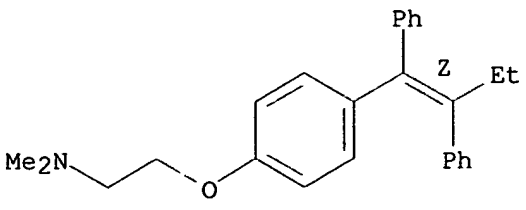
IT **10540-29-1, Tamoxifen**
RL: **BAC (Biological activity or effector, except adverse)**; **BSU (Biological study, unclassified)**; **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(methods to reduce sensitivity of endothelially-compromised vascular smooth muscle using **CLC3 chloride channel blockers** such as **tamoxifen** in relation to use of other agents)

IT **51-41-2, Norepinephrine** 7447-40-7, Potassium **chloride** (KCl), biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(vasoconstriction after endothelial damage from, normalization of; methods to reduce sensitivity of endothelially-compromised vascular smooth muscle using **CLC3 chloride channel blockers** such as **tamoxifen** in relation to use of other agents)

IT **10540-29-1, Tamoxifen**
RL: **BAC (Biological activity or effector, except adverse)**; **BSU (Biological study, unclassified)**; **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(methods to reduce sensitivity of endothelially-compromised vascular smooth muscle using **CLC3 chloride channel blockers** such as **tamoxifen** in relation to use of other agents)

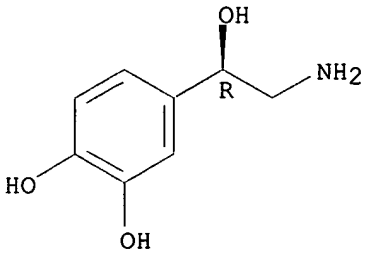
RN 10540-29-1 HCAPLUS
CN Ethanamine, 2-[4-[(1Z)-1,2-diphenyl-1-butenyl]phenoxy]-N,N-dimethyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



IT **51-41-2, Norepinephrine**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(vasoconstriction after endothelial damage from, normalization of; methods to reduce sensitivity of endothelially-compromised vascular smooth muscle using **CLC3 chloride channel blockers** such as **tamoxifen** in relation to use of other agents)
RN 51-41-2 HCAPLUS
CN 1,2-Benzenediol, 4-[(1R)-2-amino-1-hydroxyethyl]- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L79 ANSWER 4 OF 12 HCAPLUS COPYRIGHT 2003 ACS on STN
AN 2000:392207 HCAPLUS
DN 133:117961
TI Endothelium modulates anion channel-dependent aortic contractions to iodide
AU **Lamb, Fred S.**; Barna, Thomas J.
CS Department of Pediatrics, University of Iowa, Iowa City, IA, 52242, USA
SO American Journal of Physiology (2000), 278(5, Pt. 2), H1527-H1536
CODEN: AJPHAP; ISSN: 0002-9513
PB American Physiological Society
DT Journal
LA English
CC 13-6 (Mammalian Biochemistry)
AB Anion currents contribute to vascular smooth muscle (VSM) membrane potential. The substitution of extracellular **chloride** (**Cl**) with iodide (I) or bromide (Br) initially inhibited and then potentiated isometric contractile responses of rat aortic rings to **norepinephrine**. Anion substitution alone produced a small relaxation, which occurred despite a lack of active tone and minimal subsequent contraction of endothelium-intact rings (4.2 .+-. 1.2% of the response to 90 mM KCl). Endothelium-denuded rings underwent a similar initial relaxation but then contracted vigorously (I > Br). Responses to 130 mM I (93.7 .+-. 1.9% of 90 mM KCl) were inhibited by nifedipine (10-6 M), niflumic acid (10-5 M), **tamoxifen** (10-5 M), DIDS (10-4 M), and HCO3--free buffer (HEPES 10 mM) but not by bumetanide (10-5 M). Intact rings treated with N.omega.-nitro-L-arginine (10-4 M) responded weakly to I (15.5 .+-. 2.1% of 90 mM KCl), whereas Hb (10-5 M), indomethacin (10-6 M), 17-octadecynoic acid (10-5 M), and 1H-[1,2,4]oxadiazole[4,3-a]quinoxalin-1-one (10-6 M) all failed to augment the response of intact rings to I. We hypothesize that VSM takes up I primarily via an anion exchanger. Subsequent I efflux through anion **channels** having a selectivity of I > Br > **Cl** produces depolarization. In endothelium-denuded or agonist-stimulated vessels, this current is sufficient to activate voltage-dependent calcium **channels** and cause contraction. Neither nitric oxide nor prostaglandins are the primary endothelial modulator of these anion **channels**. If they are regulated by an endothelium-dependent

hyperpolarizing factor it is not a cytochrome P 450 metabolite.

ST iodide bromide anion **channel** vasoconstriction vascular smooth muscle; **chloride** calcium **channel** anion exchanger membrane depolarization iodide vasoconstriction; nitric oxide vascular endothelium iodide vasoconstriction

IT **Artery**
(aorta, endothelium; endothelium modulation of anion channel-dependent aortic contractions to iodide and bromide)

IT Transport proteins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(bicarbonate-**chloride**-exchanging; involvement of calcium and **chloride channels**, anion exchanger and membrane depolarization in anion **channel**-dependent aortic contractions to iodide and bromide)

IT Membrane potential
(biol.; involvement of calcium and **chloride channels**, anion exchanger and membrane depolarization in anion **channel**-dependent aortic contractions to iodide and bromide)

IT Polarization
(depolarization, biol.; involvement of calcium and **chloride channels**, anion exchanger and membrane depolarization in anion **channel**-dependent aortic contractions to iodide and bromide)

IT **Vasoconstriction**
Vasodilation
(endothelium modulation of anion channel-dependent aortic contractions to iodide and bromide)

IT Anion channel
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(endothelium modulation of anion channel-dependent aortic contractions to iodide and bromide)

IT **Chloride channel**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(involvement of calcium and **chloride channels**, anion exchanger and membrane depolarization in anion **channel**-dependent aortic contractions to iodide and bromide)

IT Permeability
(selective, of anion **channel** to I, Br, and **Cl**; endothelium modulation of anion **channel**-dependent aortic contractions to iodide and bromide)

IT **Blood vessel**
(**smooth muscle**; **endothelium** modulation of anion channel-dependent aortic contractions to iodide and bromide)

IT Calcium **channel**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(voltage-dependent; involvement of calcium and **chloride channels**, anion exchanger and membrane depolarization in anion **channel**-dependent aortic contractions to iodide and bromide)

IT 20461-54-5, Iodide, biological studies 24959-67-9, Bromide, biological studies
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(endothelium modulation of anion channel-dependent aortic contractions to iodide and bromide)

IT 10102-43-9, Nitric oxide, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(endothelium modulation of anion channel-dependent aortic contractions to iodide and bromide in relation to)

IT 16887-00-6, **Chloride**, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(involvement of calcium and **chloride channels**, anion exchanger and membrane depolarization in anion **channel** -dependent aortic contractions to iodide and bromide)

RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD

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L79 ANSWER 5 OF 12 HCAPLUS COPYRIGHT 2003 ACS on STN
AN **1999:782960** HCAPLUS
DN **132:384**
TI **Tamoxifen** versus placebo in the treatment of peyronie's disease
AU Teloken, Claudio; Rhoden, Ernani Luis; Grazziotin, Tulio Meyer; Da Ros, Carlos Teodosio; Sogari, Paulo Roberto; Souto, Carlos Ary Vargas
CS Department of Urology, Andrology Section, Santa Casa Hospital and Fundacao Faculdade Federal de Ciencias Medicas, Alegre, Brazil
SO Journal of Urology (Baltimore) (1999), 162(6), 2003-2005
CODEN: JOURAA; ISSN: 0022-5347

PB Lippincott Williams & Wilkins
DT Journal
LA English
CC 1-12 (**Pharmacology**)
AB Purpose: We evaluated the effects of oral **tamoxifen** and placebo in patients with Peyronie's disease. Materials and Methods: We selected 25 patients with Peyronie's disease who did not have calcified plaque for treatment in the androl. outpatient clinic. A medical history was obtained, and phys. examn., penile x-ray, penile ultrasound and pharmacol. induced erection with prostaglandin E1 were performed. Patients were randomly divided into group 1-those who received 20 mg. **tamoxifen** twice daily for 3 mo and group 2-those who received placebo for the same period. The same evaluations were done 4 mo later and results were compared. Qual. (chi-square test) and quant. (Student's t test) results were analyzed using the Yates correction factor with $p < 0.05$ considered significant. Results: Pain subsided in 66.6 and 75% of the patients treated with **tamoxifen** and placebo, resp. ($p > 0.05$). In groups 1 and 2 a redn. in the penile deformity was noticed by 46.1 and 41.7% of the patients ($p > 0.05$), and a decrease in plaque size was noticed by 30.7 and 25%, resp. On the other hand, objective measurements did not reveal any difference in plaque area or curvature angle. Conclusions: This study did not show significant improvement in pain, curvature or plaque size in patients with Peyronie's disease who were treated with **tamoxifen** compared with those treated with placebo.
ST **tamoxifen** peyronie disease sexual disorder penis
IT Sexual behavior
(disorder, penis; **tamoxifen** vs. placebo in treatment of peyronie's disease in humans)
IT **Penis**
(peyronie's disease; **tamoxifen** vs. placebo in treatment of peyronie's disease in humans)
IT **10540-29-1, Tamoxifen**
RL: **BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**tamoxifen** vs. placebo in treatment of peyronie's disease in humans)
RE.CNT 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD
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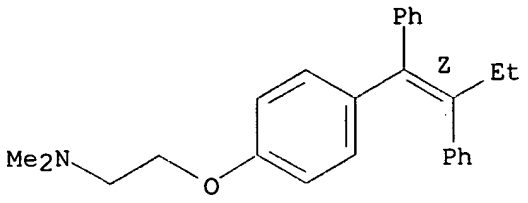
IT **10540-29-1, Tamoxifen**
RL: **BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**tamoxifen** vs. placebo in treatment of peyronie's disease in humans)
RN 10540-29-1 HCAPLUS
CN Ethanamine, 2-[4-[(1Z)-1,2-diphenyl-1-butenyl]phenoxy]-N,N-dimethyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L79 ANSWER 6 OF 12 HCAPLUS COPYRIGHT 2003 ACS on STN
AN 1998:492891 HCAPLUS
DN 129:211974
TI Chloride ion currents contribute functionally to **norepinephrine** -induced vascular contraction
AU **Lamb, Fred S.**; Barna, Thomas J.
CS Department of Pediatrics, University of Iowa, Iowa City, IA, 52242, USA
SO American Journal of Physiology (**1998**), 275(1, Pt. 2), H151-H160
CODEN: AJPHAP; ISSN: 0002-9513
PB American Physiological Society
DT Journal
LA English
CC 2-8 (Mammalian Hormones)
AB **Norepinephrine** (NE) increases **Cl**- efflux from vascular smooth muscle (VSM) cells. An increase in **Cl**- conductance produces membrane depolarization. The authors hypothesized that if **Cl**- currents are important for agonist-induced depolarization, then interfering with cellular **Cl**- handling should alter contractility. Isometric contraction of rat aortic rings was studied in a bicarbonate buffer. Substitution of extracellular **Cl**- with 130 mM methanesulfonate (MS; 8 mM **Cl**-) did not cause contraction. NE- and serotonin-induced contractions were potentiated in this low-**Cl**- buffer, whereas responses to K+, BAY K 8644, or NE in the absence of Ca2+ were unaltered. Substitution of **Cl**- with I- or Br- suppressed responses to NE. Inhibition of **Cl**- transport with bumetanide (10-5 M) or bicarbonate-free conditions (10 mM HEPES) inhibited NE- but not KCl-induced contraction. The **Cl**--**channel blockers** DIDS (10-3 M), anthracene-9-carboxylic acid (10-3 M), and niflumic acid (10-5 M) all inhibited NE-induced contraction, whereas **tamoxifen** (10-5 M) did not. Finally, disruption of sarcoplasmic reticular function with cyclopiazonic acid (10-7 M) or ryanodine (10-5 M) prevented the increase in the peak response to NE produced by low-**Cl**- buffer. The authors conclude that a **Cl**- current with a permeability sequence of I- > Br- > **Cl** - > MS is crit. to agonist-induced contraction of VSM.
ST chloride current **norepinephrine** vasoconstriction
IT Biological transport
(**channel**-mediated; **chloride** ion currents contribute functionally to **norepinephrine**-induced vascular contraction in rats)
IT **Vasoconstriction**
(chloride ion currents contribute functionally to **norepinephrine**-induced vascular contraction in rats)
IT Calcium **channel**
Chloride channel
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**chloride** ion currents contribute functionally to **norepinephrine**-induced vascular contraction in rats)
IT Biological transport
(cotransport, sodium-potassium-chloride; chloride ion currents

contribute functionally to **norepinephrine**-induced vascular contraction in rats)

IT Polarization
(depolarization, biol.; chloride ion currents contribute functionally to **norepinephrine**-induced vascular contraction in rats)

IT Endoplasmic reticulum
(sarcoplasmic reticulum; chloride ion currents contribute functionally to **norepinephrine**-induced vascular contraction in rats)

IT **Blood vessel**
(**smooth muscle**; chloride ion currents contribute functionally to **norepinephrine**-induced vascular contraction in rats)

IT 50-67-9, Serotonin, biological studies **51-41-2**, **Norepinephrine**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(chloride ion currents contribute functionally to **norepinephrine**-induced vascular contraction in rats)

IT 7440-09-7, Potassium, biological studies 7440-23-5, Sodium, biological studies 7440-70-2, Calcium, biological studies 16887-00-6, Chloride, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(chloride ion currents contribute functionally to **norepinephrine**-induced vascular contraction in rats)

IT 7440-09-7, Potassium, biological studies 7440-23-5, Sodium, biological studies 7440-70-2, Calcium, biological studies 16887-00-6, Chloride, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(transport; chloride ion currents contribute functionally to **norepinephrine**-induced vascular contraction in rats)

RE.CNT 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD

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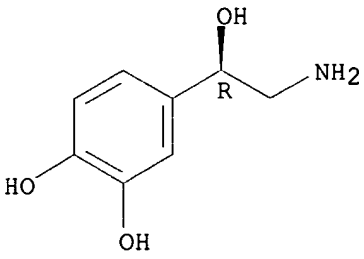
IT **51-41-2, Norepinephrine**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(chloride ion currents contribute functionally to **norepinephrine**-induced vascular contraction in rats)

RN 51-41-2 HCAPLUS
CN 1,2-Benzenediol, 4-[(1R)-2-amino-1-hydroxyethyl]- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

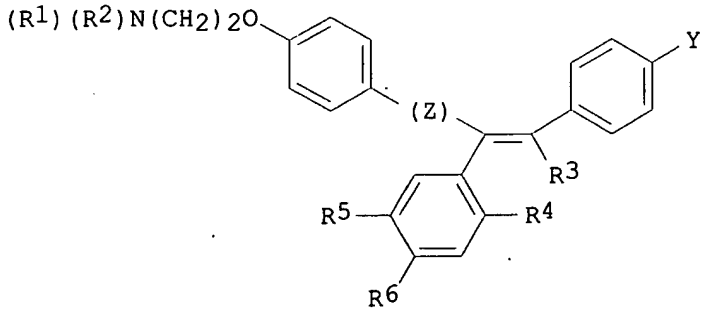


L79 ANSWER 7 OF 12 HCAPLUS COPYRIGHT 2003 ACS on STN
AN 1998:427775 HCAPLUS
DN 129:76502
TI Prevention and treatment of **cardiovascular pathologies**
IN Grainger, David J.; Metcalfe, James C.; Kunz, Lawrence L.; Schroff, Robert W.; Weissberg, Peter L.
PA NeoRx Corp., USA
SO U.S., 41 pp., Cont.-in-part of U.S. Ser. No. 242,161.
CODEN: USXXAM
DT Patent
LA English
IC ICM A61K031-445
ICS A61K031-40; A61K031-38; A61K031-135
NCL 514319000
CC 1-8 (Pharmacology)
FAN.CNT 14

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	US 5770609	A	19980623	US 1995-486334	19950607	<--
	US 5847007	A	19981208	US 1994-242161	19940512	<--
	US 5599844	A	19970204	US 1995-528810	19950915	<--
	US 5773479	A	19980630	US 1995-560808	19951121	<--
	CA 2223595	AA	19961219	CA 1996-2223595	19960607	<--
	WO 9640098	A2	19961219	WO 1996-US10211	19960607	<--
	WO 9640098	A3	19970619			

W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE,

ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS,
LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD,
SE, SG
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR,
IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA
AU 9662773 A1 19961230 AU 1996-62773 19960607 <--
EP 833624 A2 19980408 EP 1996-921577 19960607 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI
JP 11510479 T2 19990914 JP 1996-502239 19960607 <--
US 5945456 A 19990831 US 1997-965254 19971106 <--
US 6197789 B1 20010306 US 1997-973570 19971205 <--
US 6251920 B1 20010626 US 1998-82643 19980521 <--
US 6262079 B1 20010717 US 1999-306606 19990506 <--
US 2002068731 A1 20020606 US 2001-754775 20010104 <--
PRAI US 1993-11669 B2 19930128 <--
US 1993-61714 B2 19930513 <--
US 1993-62451 B2 19930513 <--
US 1994-241844 A2 19940512 <--
US 1994-242161 A2 19940512 <--
WO 1992-US8220 B2 19920925 <--
US 1994-300357 A1 19940902 <--
US 1995-476735 A 19950607 <--
US 1995-477393 A 19950607 <--
US 1995-478936 A 19950607 <--
US 1995-486334 A 19950607 <--
US 1995-560808 A1 19951121 <--
WO 1996-US10211 W 19960607 <--
US 1997-973570 A1 19971205 <--
US 1998-82643 A1 19980521 <--
OS MARPAT 129:76502
GI



I

AB A method for treating or preventing cardiovascular pathologies by administering I, wherein Z is CO or a covalent bond; Y is H or O(C1-C4)alkyl, R1 and R2 are individually (C1-C4)alkyl or together with N are a satd. heterocyclic group, R3 is Et or chloroethyl, R4 is H or together with R3 is -CH2CH2- or -S-, R5 is I, O(C1-C4)alkyl or H and R6 is I, O(C1-C4)alkyl or H with the proviso that when R4, R5, and R6 are H, R3 is not ethyl; or a pharmaceutically acceptable salt thereof, effective to activate or stimulate prodn. of TGF-beta to treat and/or prevent conditions such as atherosclerosis, thrombosis, myocardial infarction, and stroke is provided. Useful compds. include idoxifene and salts thereof. Further provided is a method for identifying a compd. that is a TGF-beta activator or prodn. stimulator is provided. Another embodiment of the invention is an assay or kit to det. TGF-beta in vitro. Also provided is a therapeutic method comprising inhibiting smooth muscle cell proliferation assocd. with procedural vascular trauma employing the

administration of **tamoxifen** or structural analogs thereof, including I. Some of the examples include impact of **tamoxifen** on vascular smooth muscle cells (VSMC) and the relationship to TGF-.beta. prodn. and activation and heparin effect on VSMC proliferation and differentiation.

ST TGFb prodn cardiovascular pathol

IT **Blood vessel**
Cell proliferation
(TGF-.beta. activators or prodn. stimulators in prevention and treatment of cardiovascular pathologies)

IT Cardiovascular system
(disease; TGF-.beta. activators or prodn. stimulators in prevention and treatment of cardiovascular pathologies)

IT **Blood vessel**
(**smooth muscle**; TGF-.beta. activators or prodn. stimulators in prevention and treatment of cardiovascular pathologies)

IT Transforming growth factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(.beta.-; TGF-.beta. activators or prodn. stimulators in prevention and treatment of cardiovascular pathologies)

IT 89778-26-7, Toremifene 116057-66-0 116057-75-1, Idoxifene 116057-76-2
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(TGF-.beta. activators or prodn. stimulators in prevention and treatment of cardiovascular pathologies)

RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

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(6) Anon; EP 0374044 B1 1990 HCAPLUS
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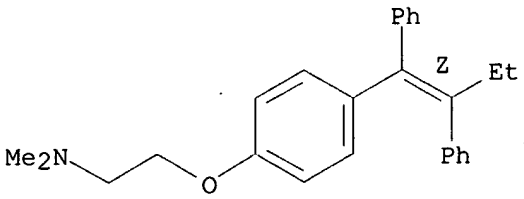
L79 ANSWER 8 OF 12 HCAPLUS COPYRIGHT 2003 ACS on STN
AN **1997:404608** HCAPLUS
DN **127:60325**
TI Coronary heart disease mortality and adjuvant **tamoxifen** therapy
AU Costantino, Joseph P.; Kuller, Lewis H.; Ives, Diane G.; Fisher, Bernard; Dignam, James
CS Department of Biostatistics, University of Pittsburgh, Pittsburgh, PA, 15261, USA
SO Journal of the National Cancer Institute (**1997**), 89(11), 776-782
CODEN: JNCIEQ; ISSN: 0027-8874
PB Oxford University Press
DT Journal
LA English
CC 1-6 (**Pharmacology**)
AB Data from randomized clin. trials in Scotland and Sweden testing the efficacy of **tamoxifen** therapy in patients with breast cancer have suggested that the drug may also reduce the risk of coronary heart disease. In view of these findings, the authors examd. mortality from coronary heart disease among patients with early stage breast cancer who were enrolled in the National Surgical Adjuvant Breast and Bowel Project B-14 trial of **tamoxifen** therapy. Deaths occurring among women who were randomly assigned to 5 yr of either **tamoxifen** or placebo in the first phase of the B-14 trial were reviewed to det. the cause. Three categories of heart disease-related death were defined: (1) death from a definite fatal myocardial infarction, (2) death from definite fatal coronary heart disease/possible myocardial infarction, and (3) death from possible fatal coronary heart disease. Comparisons of the findings by treatment group were made on the basis of av. annual hazard (i.e., death) rates and the corresponding relative hazard of death. The av. annual death rate from coronary heart disease was lower for patients who received **tamoxifen** than for patients who received placebo, but the difference was not statistically significant. There were eight definite heart-related deaths (i.e., definite fatal myocardial infarction or definite fatal coronary heart disease/possible myocardial infarction) among the patients who received **tamoxifen**, yielding an av. annual rate of 0.62 per 1000 patients. There were 12 definite heart-related deaths among the patients who received placebo, yielding an av. annual rate of 0.94 per 1000. The corresponding relative hazard of death from definite fatal heart disease (**tamoxifen** vs. placebo) was 0.66 (95% confidence interval = 0.27-1.61). Eleven deaths in the **tamoxifen** group and 10 deaths in the placebo group were classified as possible cases of fatal coronary heart disease. When these cases and the definite cases were considered together, the av. annual death rate for the patients who received **tamoxifen** was 1.48 per 1000, and the rate for the patients who received placebo was 1.73 per 1000. The corresponding relative hazard of death was 0.85 (95% confidence interval = 0.46-1.58). The findings from the B-14 trial are consistent with the findings from the Scottish and the Swedish trials, suggesting that **tamoxifen** treatment reduces coronary heart disease among patients with breast cancer. Continued follow-up of the patients in these trials and in ongoing prevention trials is needed to accumulate enough data so that reliable conclusions can be drawn about the benefits of **tamoxifen** in preventing heart disease.
ST coronary heart disease **tamoxifen** breast cancer; cancer antitumor

IT **Artery, disease**
(coronary; coronary heart disease mortality and adjuvant **tamoxifen** therapy in humans)
IT Antitumor agents
(mammary gland; coronary heart disease mortality and adjuvant **tamoxifen** therapy in humans)
IT Mammary gland
(neoplasm, inhibitors; coronary heart disease mortality and adjuvant **tamoxifen** therapy in humans)
IT **10540-29-1, Tamoxifen**
RL: ADV (Adverse effect, including toxicity); **BAC (Biological activity or effector, except adverse)**; **BSU (Biological study, unclassified)**; **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(coronary heart disease mortality and adjuvant **tamoxifen** therapy in humans)
IT **10540-29-1, Tamoxifen**
RL: ADV (Adverse effect, including toxicity); **BAC (Biological activity or effector, except adverse)**; **BSU (Biological study, unclassified)**; **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(coronary heart disease mortality and adjuvant **tamoxifen** therapy in humans)
RN 10540-29-1 HCAPLUS
CN Ethanamine, 2-[4-[(1Z)-1,2-diphenyl-1-butenyl]phenoxy]-N,N-dimethyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L79 ANSWER 9 OF 12 HCAPLUS COPYRIGHT 2003 ACS on STN
AN **1997:134850** HCAPLUS
DN **126:139887**
TI Prevention and treatment of cardiovascular pathologies with **tamoxifen** analogs
IN Grainger, David J.; Metcalfe, James C.; Kunz, Lawrence L.; Kemp, Paul R.; Schroff, Robert W.; Weissberg, Peter L.
PA Neorx Corporation, USA; Grainger, David J.; Metcalfe, James C.; Kunz, Lawrence L.; Kemp, Paul R.; Schroff, Robert W.; Weissberg, Peter L.
SO PCT Int. Appl., 130 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K031-135
CC 1-8 (**Pharmacology**)
Section cross-reference(s): **63**
FAN.CNT 14

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9640098	A2	19961219	WO 1996-US10211	19960607 <--
WO 9640098	A3	19970619		

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SE, SG
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA

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US 5770609	A	19980623	US 1995-486334	19950607	<--
US 6395494	B1	20020528	US 1995-477393	19950607	<--
AU 9662773	A1	19961230	AU 1996-62773	19960607	<--
EP 833624	A2	19980408	EP 1996-921577	19960607	<--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI					
JP 11510479	T2	19990914	JP 1996-502239	19960607	<--
US 6197789	B1	20010306	US 1997-973570	19971205	<--
US 2002068731	A1	20020606	US 2001-754775	20010104	<--

PRAI US 1995-476735	A	19950607	<--	
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US 1995-486334	A	19950607	<--	
US 1993-11669	B2	19930128	<--	
US 1993-61714	B2	19930513	<--	
US 1993-62451	B2	19930513	<--	
US 1994-241844	A2	19940512	<--	
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US 1997-973570	A1	19971205	<--	

OS MARPAT 126:139887

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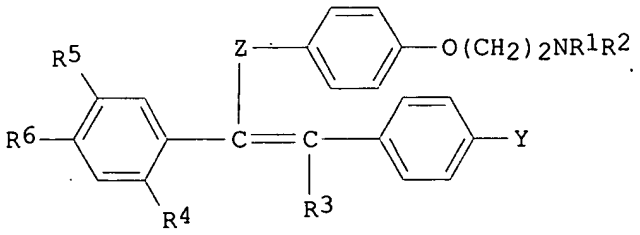


I

AB A method for treating or preventing cardiovascular pathologies comprises administering a compd. I (Z = C:O, covalent bond; Y = H, O(C1-4)alkyl; R1, R2 = (C1-4)alkyl, together with N satd. heterocyclic group; R3 = Et, chloroethyl; R4 = H; R5 = I, O(C1-4)alkyl, H; R6 = I, O(C1-C4)alkyl, H, with the proviso that when R4, R5, and R6 are H, R3 is not ethyl) or a pharmaceutically acceptable salt thereof, effective to elevate the level of TGF-.beta. to treat and/or prevent conditions such as atherosclerosis, thrombosis, myocardial infarction, and stroke is provided. Useful compds. include idoxifene, raloxifene, toremifene, droloxifene or salts thereof. A method for identifying an agent that elevates the level of TGF-beta and an assay or kit to det. TGF-.beta. based on anti-TGF-.beta.-antibodies are also provided. **Tamoxifen** (1.1 mg/kg/day) strongly inhibited the formation of lipid lesions induced by a high fat diet in mice. The major effect of **tamoxifen** in mice was to elevate TGF-.beta. in aortic wall and in circulation.

ST **tamoxifen** analog cardiovascular disease; transforming growth factor beta **tamoxifen** analog

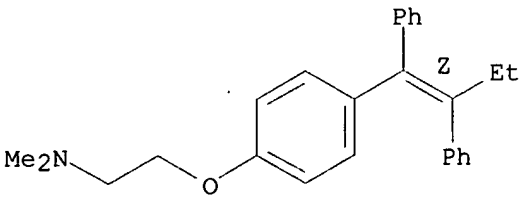
IT mRNA
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(TGF-.beta., prodn. of, increase of; **tamoxifen** analogs for prevention and treatment of cardiovascular diseases)

IT DNA
RL: BSU (Biological study, unclassified); BIOL (Biological study)

(adducts; **tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT Antiarteriosclerotics
(antiatherosclerotics; **tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT Medical goods
(catheters, for drug delivery to arterial lesion; **tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT **Artery**
Artery
(coronary, angioplasty, drug delivery in; **tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT Eye, disease
(diabetic retinopathy; **tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT Heart, disease
(infarction, therapeutic agents; **tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT Heart, disease
(infarction; **tamoxifen** analogs for prevention of cardiovascular diseases)
IT **Blood vessel, disease**
(**injury**; **tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT **Artery, disease**
(lesions, drug delivery in; **tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT Antibodies
RL: ARG (Analytical reagent use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(monoclonal; **tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT Proliferation inhibition
(proliferation inhibitors, vascular smooth muscle; **tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT **Blood vessel**
(**smooth muscle**, proliferation of, inhibition of; **tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT Brain, disease
(stroke; **tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT **Blood vessel**
Blood vessel
(**surgery**; **tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT Drug delivery systems
(sustained-release; **tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT Anticoagulants
Transplant and Transplantation
(**tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT Antibodies
RL: ARG (Analytical reagent use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(**tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT **Atherosclerosis**
Thrombosis
(**tamoxifen** analogs for prevention of cardiovascular diseases)
IT Cell division
(vascular smooth muscle, inhibition of; **tamoxifen** analogs for

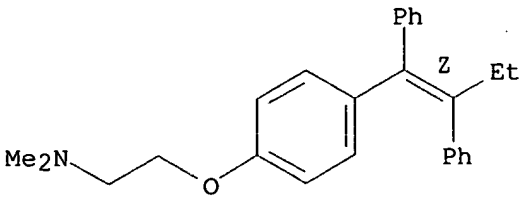
prevention and treatment of cardiovascular diseases)
IT Surgery
Surgery
(vascular; **tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT Transforming growth factors
RL: ANT (Analyte); BOC (Biological occurrence); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study); OCCU (Occurrence)
(.beta.-; **tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT **10540-29-1, Tamoxifen 10540-29-1D, Tamoxifen**, analogs 82413-20-5, Droloxifene 84449-90-1, Raloxifene 89778-26-7, Toremifene 116057-66-0 116057-75-1, Idoxifene 116057-76-2
RL: **BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
IT **10540-29-1, Tamoxifen 10540-29-1D, Tamoxifen**, analogs
RL: **BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**tamoxifen** analogs for prevention and treatment of cardiovascular diseases)
RN 10540-29-1 HCAPLUS
CN Ethanamine, 2-[4-[(1Z)-1,2-diphenyl-1-butenyl]phenoxy]-N,N-dimethyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 10540-29-1 HCAPLUS
CN Ethanamine, 2-[4-[(1Z)-1,2-diphenyl-1-butenyl]phenoxy]-N,N-dimethyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

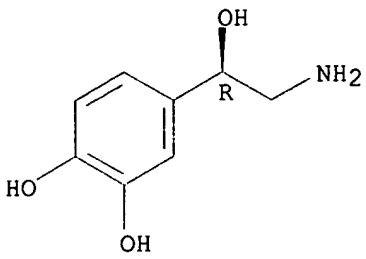


L79 ANSWER 10 OF 12 HCAPLUS COPYRIGHT 2003 ACS on STN
AN 1996:126984 HCAPLUS
DN 124:165616
TI Effect of gender and sex steroids on the contractile response of canine coronary and renal blood vessels

AU Karanian, John W.; Ramwell, Peter W.
CS Lab. Membrane Biochem. Biophys., NIAAA, Washington, DC, USA
SO Journal of Cardiovascular Pharmacology (**1996**), 27(3), 312-19
CODEN: JCPCDT; ISSN: 0160-2446
PB Lippincott-Raven
DT Journal
LA English
CC 2-4 (Mammalian Hormones)
AB The effect of gender, gonadal steroids, and antiandrogen/antiestrogen-treatment on the isotonic response of isolated prepns. of the left anterior descending coronary artery (LAD), left circumflex coronary artery (LCX), and renal artery and vein of sexually mature dogs was investigated. The max. isotonic response of the coronary and renal vasculature to the thromboxane A2 (TXA2)-mimetic U46619 was significantly greater, and the EC50 value was significantly lower in males as compared with females. Moreover, similar gender differences in the contractile response of the coronary vasculature to **norepinephrine** were obsd. Pretreatment of male dogs with the antiandrogens flutamide or cyproterone acetate reduced the max. contractile response of the LAD to the TXA2-mimetic. Pretreatment of female dogs with testosterone resulted in an increase in both the max. contractile response and EC50 value to U46619. Antiestrogen treatment of female dogs with **tamoxifen** was assocd. with an increase in the max. contractile response of the LAD to U46619. Estrogen pretreatment of male dogs decreased both the max. contractile response and the EC50 value to U46619. Therefore, there is a sex difference in LAD and LCX contractile responses to both U46619 and **norepinephrine**. These results suggest that smooth muscle reactivity of dog coronary artery to the TXA2-mimetic U46619 may be susceptible to regulation by both androgens and estrogens. The obsd. gender differences in the catecholamine response may be similarly altered by changes in the hormonal milieu.
ST blood vessel heart kidney contraction gender; TXA2 heart kidney vessel sex steroid; **norepinephrine** heart kidney vessel sex steroid; androgen heart kidney vessel contraction; estrogen heart kidney vessel contraction
IT Sex
(gender and sex steroids effects on contractile response of canine coronary and renal blood vessels to **norepinephrine** and TXA2)
IT Androgens
Estrogens
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(gender and sex steroids effects on contractile response of canine coronary and renal blood vessels to **norepinephrine** and TXA2)
IT **Artery**
(coronary, gender and sex steroids effects on contractile response of canine coronary and renal blood vessels to **norepinephrine** and TXA2)
IT **Artery**
Vein
(renal, gender and sex steroids effects on contractile response of canine coronary and renal blood vessels to **norepinephrine** and TXA2)
IT **51-41-2, Norepinephrine** 58-22-0, Testosterone
313-06-4, Estradiol cypionate 427-51-0, Cyproterone acetate
10540-29-1, Tamoxifen 13311-84-7, Flutamide
56985-40-1, U-46619
RL: **BAC (Biological activity or effector, except adverse)**; **BSU (Biological study, unclassified)**; BIOL (Biological study)
(gender and sex steroids effects on contractile response of canine coronary and renal blood vessels to **norepinephrine** and TXA2)
IT **51-41-2, Norepinephrine 10540-29-1, Tamoxifen**

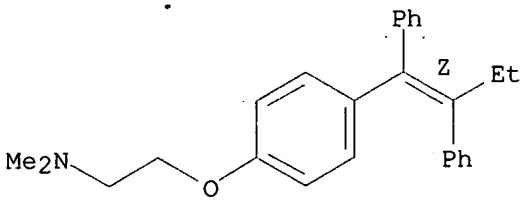
RL: **BAC (Biological activity or effector, except adverse)**; **BSU (Biological study, unclassified)**; BIOL (Biological study)
(gender and sex steroids effects on contractile response of canine coronary and renal blood vessels to **norepinephrine** and TXA2)
RN 51-41-2 HCAPLUS
CN 1,2-Benzenediol, 4-[(1R)-2-amino-1-hydroxyethyl]- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



RN 10540-29-1 HCAPLUS
CN Ethanamine, 2-[4-[(1Z)-1,2-diphenyl-1-butenyl]phenoxy]-N,N-dimethyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



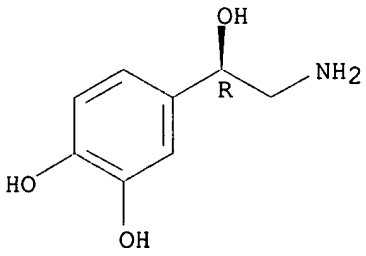
L79 ANSWER 11 OF 12 HCAPLUS COPYRIGHT 2003 ACS on STN
AN **1996:13286** HCAPLUS
DN **124:45720**
TI Method of treating peripheral vasoconstriction with **tamoxifen** citrate
IN Stromberg, Brent V.
PA USA
SO U.S., 4 pp.
CODEN: USXXAM
DT Patent
LA English
IC ICM A61K031-135
NCL 514648000
CC 1-8 (**Pharmacology**)
Section cross-reference(s): 2
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 5470883	A	19951128	US 1994-247771	19940523 <--
PRAI US 1994-247771		19940523 <--		

AB A method of modifying peripheral vasoconstriction comprises administering a pharmacol. acceptable dose of a **tamoxifen** salt, e.g. **tamoxifen** citrate.
ST **tamoxifen** citrate peripheral vasoconstriction treatment
IT Adrenergic agonists
Blood vessel
(**tamoxifen** salts for treating **peripheral**

vasoconstriction)
IT **51-41-2, Norepinephrine** 51-43-4, Epinephrine
51-61-6, Dopamine, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(**tamoxifen** salts for treating peripheral vasoconstriction)
IT **54965-24-1, Tamoxifen** citrate
RL: **BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**tamoxifen** salts for treating peripheral vasoconstriction)
IT **10540-29-1D, Tamoxifen,** salts
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**tamoxifen** salts for treating peripheral vasoconstriction)
IT **51-41-2, Norepinephrine**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(**tamoxifen** salts for treating peripheral vasoconstriction)
RN 51-41-2 HCAPLUS
CN 1,2-Benzenediol, 4-[(1R)-2-amino-1-hydroxyethyl]- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

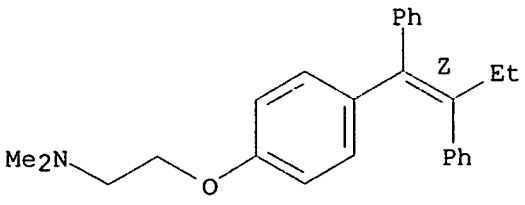


IT **54965-24-1, Tamoxifen** citrate
RL: **BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**tamoxifen** salts for treating peripheral vasoconstriction)
RN 54965-24-1 HCAPLUS
CN Ethanamine, 2-[4-[(1Z)-1,2-diphenyl-1-butenyl]phenoxy]-N,N-dimethyl-, 2-hydroxy-1,2,3-propanetricarboxylate (1:1) (9CI) (CA INDEX NAME)

CM 1

CRN 10540-29-1
CMF C26 H29 N O

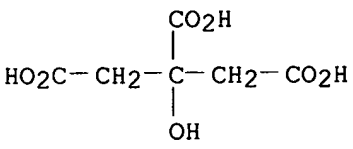
Double bond geometry as shown.



CM 2

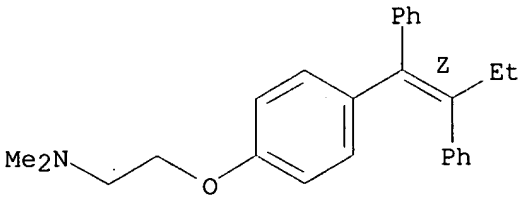
CRN 77-92-9

CMF C6 H8 O7



IT **10540-29-1D, Tamoxifen,** salts
RL: **THU (Therapeutic use);** BIOL (Biological study); USES (Uses)
(tamoxifen salts for treating peripheral vasoconstriction)
RN 10540-29-1 HCAPLUS
CN Ethanamine, 2-[4-[(1Z)-1,2-diphenyl-1-butenyl]phenoxy]-N,N-dimethyl- (9CI)
(CA INDEX NAME)

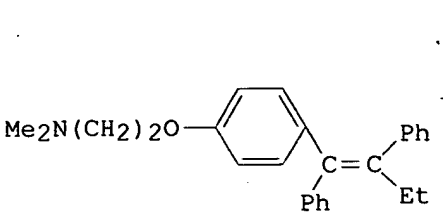
Double bond geometry as shown.



L79 ANSWER 12 OF 12 HCAPLUS COPYRIGHT 2003 ACS on STN
AN **1985:154801** HCAPLUS
DN **102:154801**
TI Prophylaxis and therapy for coronary heart diseases by lowering the estrogen level
IN Schulze, Paul Eberhard; Kerb, Ulrich
PA Schering A.-G. , Fed. Rep. Ger.
SO Ger. Offen., 9 pp.
CODEN: GWXXBX
DT Patent
LA German
IC ICM A61K045-06
ICS A61K031-135; A61K031-12; A61K031-40; A61K031-565
CC 63-6 (**Pharmaceuticals**)
Section cross-reference(s): 2
FAN.CNT 1

	PATENT NO.	KIND	DATE		APPLICATION NO.	DATE	
PI	DE 3323321	A1	19850103		DE 1983-3323321	19830624	<--
	WO 8500107	A1	19850117		WO 1984-DE137	19840619	<--
	W: JP, US						
	RW: DE, FR, GB, NL						
	JP 60501656	T2	19851003		JP 1984-502536	19840619	<--
	EP 179062	A1	19860430		EP 1984-902507	19840619	<--
	R: DE, FR, GB, NL						
PRAI	DE 1983-3323321		19830624	<--			
	WO 1984-DE137		19840619	<--			

GI



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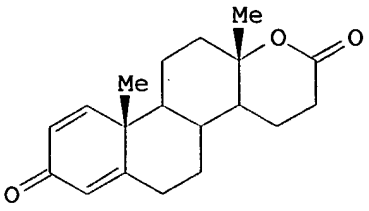


II

AB Coronary heart disease in men is treated by administration of an antiestrogen, such as **tamoxifen** (I) [**10540-29-1**], or an aromatase inhibitor, such as testololactone (II) [4416-57-3], in daily doses of 10-200 or 50-1000 mg, resp. Tablets were prepd. contg. II 100, lactose 80.5, cornstarch 39.5, PVP 2.5, Aerosil 2.0, and Mg stearate 0.5 mg.

ST coronary heart disease **tamoxifen** testololactone

IT **Heart, disease or disorder**
(**coronary**, treatment of, with **tamoxifen** and testololactone pharmaceuticals in men)

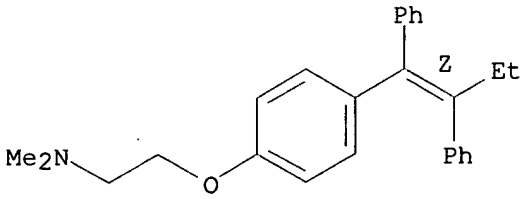
IT 4416-57-3 **10540-29-1**
RL: BIOL (Biological study)
(pharmaceuticals, for coronary heart disease treatment, in men)

IT **10540-29-1**
RL: BIOL (Biological study)
(pharmaceuticals, for coronary heart disease treatment, in men)

RN 10540-29-1 HCAPLUS

CN Ethanamine, 2-[4-[(1Z)-1,2-diphenyl-1-butenyl]phenoxy]-N,N-dimethyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



=> sel hit rn 179
E40 THROUGH E42 ASSIGNED

=> fil reg
FILE 'REGISTRY' ENTERED AT 10:48:50 ON 27 AUG 2003
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
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Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 25 AUG 2003 HIGHEST RN 573649-48-6
DICTIONARY FILE UPDATES: 25 AUG 2003 HIGHEST RN 573649-48-6

TSCA INFORMATION NOW CURRENT THROUGH JANUARY 6, 2003

Please note that search-term pricing does apply when

conducting SmartSELECT searches.

Crossover limits have been increased. See HELP CROSSOVER for details.

Experimental and calculated property data are now available. See HELP PROPERTIES for more information. See STNote 27, Searching Properties in the CAS Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

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=> s e40-e42
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               (10540-29-1/RN)
             1 51-41-2/BI
               (51-41-2/RN)
             1 54965-24-1/BI
               (54965-24-1/RN)
L80          3 (10540-29-1/BI OR 51-41-2/BI OR 54965-24-1/BI)

=> d ide can tot

L80  ANSWER 1 OF 3  REGISTRY  COPYRIGHT 2003 ACS on STN
RN   54965-24-1  REGISTRY
CN   Ethanamine, 2-[4-[(1Z)-1,2-diphenyl-1-butenyl]phenoxy]-N,N-dimethyl-,
     2-hydroxy-1,2,3-propanetricarboxylate (1:1) (9CI)  (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   Ethanamine, 2-[4-(1,2-diphenyl-1-butenyl)phenoxy]-N,N-dimethyl-, (Z)-,
     2-hydroxy-1,2,3-propanetricarboxylate (1:1)
OTHER NAMES:
CN   I.C.I.46474 citrate
CN   ICI 46474
CN   Kessar
CN   Noltam
CN   Nolvadex
CN   Nourytam
CN   Tamofen
CN   Tamoplex
CN   Tamox-Puren
CN   Tamoxifen citrate
CN   TMX
CN   Tomaxasta
CN   Z-Tamoxifen citrate
CN   Zemide
FS   STEREOSEARCH
DR   7244-97-5
MF   C26 H29 N O . C6 H8 O7
CI   COM
LC   STN Files:   ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS,
       BIOTECHNO, CA, CAOLD, CAPLUS, CBNB, CEN, CHEMCATS, CHEMLIST, CIN,
       CSCHEM, DIOGENES, DRUGPAT, EMBASE, HSDB*, IPA, MRCK*, MSDS-OHS,
       PHARMASEARCH, PIRA, PROMT, RTECS*, TOXCENTER, ULIDAT, USAN, USPATFULL
         (*File contains numerically searchable property data)
     Other Sources:   EINECS**
         (**Enter CHEMLIST File for up-to-date regulatory information)

     CM   1

     CRN  10540-29-1
     CMF  C26 H29 N O

Double bond geometry as shown.
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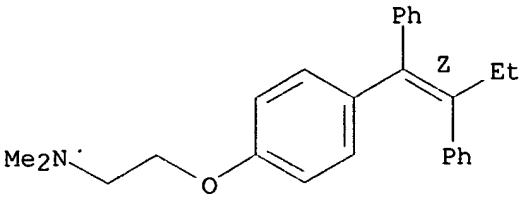
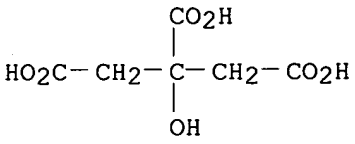


CM 2

CRN 77-92-9
CMF C6 H8 O7



219 REFERENCES IN FILE CA (1937 TO DATE)
219 REFERENCES IN FILE CAPLUS (1937 TO DATE)

REFERENCE 1: 139:101145

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REFERENCE 3: 139:101141

REFERENCE 4: 139:79130

REFERENCE 5: 139:63529

REFERENCE 6: 139:47180

REFERENCE 7: 139:30831

REFERENCE 8: 139:12264

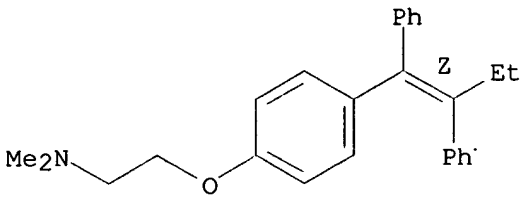
REFERENCE 9: 138:406952

REFERENCE 10: 138:395618

L80 ANSWER 2 OF 3 REGISTRY COPYRIGHT 2003 ACS on STN
RN **10540-29-1** REGISTRY
CN Ethanamine, 2-[4-[(1Z)-1,2-diphenyl-1-butenyl]phenoxy]-N,N-dimethyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Ethanamine, 2-[4-(1,2-diphenyl-1-butenyl)phenoxy]-N,N-dimethyl-, (Z)-
CN Ethylamine, 2-[p-(1,2-diphenyl-1-butenyl)phenoxy]-N,N-dimethyl-, (Z)- (8CI)
OTHER NAMES:
CN ICI 47699
CN Mammaton
CN Tamoxifen
CN trans-Tamoxifen
CN Z-Tamoxifen
FS STEREOSEARCH
MF C26 H29 N O

CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DIOGENES, DRUGNL, DRUGPAT, DRUGU, EMBASE, HSDB*, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PHAR, PHARMASEARCH, PROMT, RTECS*, SPECINFO, TOXCENTER, ULIDAT, USAN, USPAT2, USPATFULL, VETU
(*File contains numerically searchable property data)
Other Sources: EINECS**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)

Double bond geometry as shown.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

5019 REFERENCES IN FILE CA (1937 TO DATE)
136 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
5032 REFERENCES IN FILE CAPLUS (1937 TO DATE)

REFERENCE 1: 139:131176

REFERENCE 2: 139:129670

REFERENCE 3: 139:129225

REFERENCE 4: 139:128591

REFERENCE 5: 139:128133

REFERENCE 6: 139:127969

REFERENCE 7: 139:127663

REFERENCE 8: 139:127602

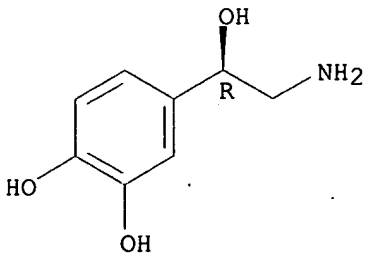
REFERENCE 9: 139:127600

REFERENCE 10: 139:127537

L80 ANSWER 3 OF 3 REGISTRY COPYRIGHT 2003 ACS on STN
RN **51-41-2** REGISTRY
CN 1,2-Benzenediol, 4-[(1R)-2-amino-1-hydroxyethyl]- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 1,2-Benzenediol, 4-(2-amino-1-hydroxyethyl)-, (R)-
CN Benzyl alcohol, .alpha.-(aminomethyl)-3,4-dihydroxy-, (-)- (8CI)
OTHER NAMES:
CN (-)-.alpha.-(Aminomethyl)protocatechuyl alcohol
CN (-)-Arterenol
CN (-)-Noradrenaline
CN (-)-Norepinephrine
CN (R)-(-)-Norepinephrine

CN (R)-Noradrenaline
CN (R)-Norepinephrine
CN Adrenor
CN Aktamin
CN Arterenol
CN l-.alpha.-(Aminomethyl)-3,4-dihydroxybenzyl alcohol
CN l-1-(3,4-Dihydroxyphenyl)-2-aminoethanol
CN l-2-Amino-1-(3,4-dihydroxyphenyl)ethanol
CN l-3,4-Dihydroxyphenylethanolamine
CN l-Arterenol
CN L-Noradrenaline
CN l-Noradrenaline
CN l-Norepinephrine
CN Levarterenol
CN Levoarterenol
CN Levonor
CN Levonoradrenaline
CN Levonorepinephrine
CN Levophed
CN Nor-Epirenan
CN Noradrenalin
CN Noradrenaline
CN Norartrinal
CN Norepinephrine
CN Norepirenamine
CN Sympathin E
FS STEREOSEARCH
DR 66197-73-7, 4899-05-2
MF C8 H11 N O3
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DIOGENES, DRUGU, EMBASE, HODOC*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PROMT, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, USAN, USPAT2, USPATFULL, VETU
(*File contains numerically searchable property data)
Other Sources: EINECS**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

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59153 REFERENCES IN FILE CAPLUS (1937 TO DATE)
366 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 139:139915

REFERENCE 2: 139:133463

REFERENCE 3: 139:132202

REFERENCE 4: 139:131966

REFERENCE 5: 139:131926

REFERENCE 6: 139:131869

REFERENCE 7: 139:131668

REFERENCE 8: 139:131383

REFERENCE 9: 139:131361

REFERENCE 10: 139:131298

=> fil medline
FILE 'MEDLINE' ENTERED AT 11:02:24 ON 27 AUG 2003

FILE LAST UPDATED: 26 AUG 2003 (20030826/UP). FILE COVERS 1958 TO DATE.

On April 13, 2003, MEDLINE was reloaded. See HELP RLOAD for details.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2003 vocabulary. See http://www.nlm.nih.gov/mesh/changes2003.html for a description on changes.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> d l103 all tot

L103 ANSWER 1 OF 4 MEDLINE on STN
AN **2000108129** MEDLINE
DN **20108129** PubMed ID: **10643517**
TI New aspects in Peyronie's disease--a mini-review.
AU Schroeder-Printzen I; Hauck E W; Weidner W
CS Department of Urology, Justus Liebig University, Giessen, Germany.
SO ANDROLOGIA, **(1999)** 31 Suppl 1 31-5. Ref: 28
Journal code: 0423506. ISSN: 0303-4569.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200002
ED Entered STN: 20000218
Last Updated on STN: 20000218
Entered Medline: 20000210
AB There have been several advances in elucidation of the pathogenesis of Peyronie's disease. Transforming growth factor beta 1 seems to play a major role in this disease, while the importance of penile trauma is a matter of debate. High-resolution ultrasound sonography is the method of choice in detecting penile plaques, while magnetic resonance imaging is useful in the evaluation of actively inflamed plaques. There are still differences of opinion on the best drug therapy in noncalcified plaques. The results on **tamoxifen** or interferon therapy vary between useless and useful. Potassium-para-aminobenzoate seems to have a significant effect in decreasing plaque size and deviation angle. The

operative strategy for big plaques or complex deviation has changed to the 'small incision' graft, leading to far lower post-operative impotence rates. Iontophoresis seems to be worthy of further trials, while the results of extracorporal shock wave therapy have to be discussed critically.

CT Check Tags: Human; Male
Impotence: ET, etiology
***Penile Induration**
Penile Induration: DI, diagnosis
Penile Induration: ET, etiology
Penile Induration: TH, therapy

L103 ANSWER 2 OF 4 MEDLINE on STN
AN **2000034844** MEDLINE
DN **20034844** PubMed ID: **10569556**
TI **Tamoxifen** versus placebo in the treatment of Peyronie's disease.
AU Teloken C; Rhoden E L; Grazziotin T M; Ros C T; Sogari P R; Souto C A
CS Department of Urology, Santa Casa Hospital and Fundacao Faculdade Federal de Ciencias Medicas, Porto Alegre, Brazil.
SO JOURNAL OF UROLOGY, **(1999 Dec)** 162 (6) 2003-5.
Journal code: 0376374. ISSN: 0022-5347.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200001
ED Entered STN: 20000114
Last Updated on STN: 20000114
Entered Medline: 20000106
AB PURPOSE: We evaluated the effects of oral **tamoxifen** and placebo in patients with Peyronie's disease. MATERIALS AND METHODS: We selected 25 patients with Peyronie's disease who did not have calcified plaque for treatment in the andrology outpatient clinic. A medical history was obtained, and physical examination, penile x-ray, penile ultrasound and pharmacologically induced erection with prostaglandin E1 were performed. Patients were randomly divided into group 1--those who received 20 mg. **tamoxifen** twice daily for 3 months and group 2--those who received placebo for the same period. The same evaluations were done 4 months later and results were compared. Qualitative (chi-square test) and quantitative (Student's t test) results were analyzed using the Yates correction factor with $p < 0.05$ considered significant. RESULTS: Pain subsided in 66.6 and 75% of the patients treated with **tamoxifen** and placebo, respectively ($p > 0.05$). In groups 1 and 2 a reduction in the penile deformity was noticed by 46.1 and 41.7% of the patients ($p > 0.05$), and a decrease in plaque size was noticed by 30.7 and 25%, respectively. On the other hand, objective measurements did not reveal any difference in plaque area or curvature angle. CONCLUSIONS: This study did not show significant improvement in pain, curvature or plaque size in patients with Peyronie's disease who were treated with **tamoxifen** compared with those treated with placebo.
CT Check Tags: Human; Male
Administration, Oral
Aged
Middle Age
***Penile Induration: DT, drug therapy**
***Tamoxifen: AD, administration & dosage**
RN **10540-29-1 (Tamoxifen)**

L103 ANSWER 3 OF 4 MEDLINE on STN
AN **1998138816** MEDLINE
DN **98138816** PubMed ID: **9478329**

TI The effect of **tamoxifen** on the neonatal development of rat glans penis.
AU Deveci E; Onen A; Tacar O; Yildirim A
CS Department of Histology and Embryology, Medical Faculty, University of Dicle, Diyarbakir, Turkiye.
SO CLINICAL AND EXPERIMENTAL OBSTETRICS AND GYNECOLOGY, **(1997)** 24 (4) 237-9.
Journal code: 7802110. ISSN: 0390-6663.
CY Italy
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199803
ED Entered STN: 19980407
Last Updated on STN: 19980407
Entered Medline: 19980326
AB From the first day of birth to the fifth day, daily subcutaneous 100 micrograms **tamoxifen** (Tx) was injected into new-born male rats. The penises that were taken totally were fixated in 10% formaline, and then they were put in paraffin inclusion. The paraffin sections were stained with Hematoxylen-Eosin, Verhoeff and Triple on days 7, 14, 21, 28, 35 and 60. The alterations in the development of glans penis construction were examined. We found that in the glans penis of animals which were given Tx, from the 21st day, the epidermal projections were erased slowly and on the 60th day the epidermal projections and keratinisation completely ceased altogether. As a result, the development of epidermal projections in rats which were given **tamoxifen** in the neonatal period were hindered.
CT Check Tags: Animal; Male
*Animals, Newborn
Atrophy
*Epidermis: DE, drug effects
*Epidermis: GD, growth & development
Epidermis: PA, pathology
Epithelium: DE, drug effects
Epithelium: PA, pathology
*Estrogen Antagonists: PD, pharmacology
Injections, Subcutaneous
Keratin: ME, metabolism
***Penis: DE, drug effects**
***Penis: GD, growth & development**
Rats
Tamoxifen: AD, administration & dosage
***Tamoxifen: PD, pharmacology**
RN **10540-29-1 (Tamoxifen)**; 68238-35-7 (Keratin)
CN 0 (Estrogen Antagonists)

L103 ANSWER 4 OF 4 MEDLINE on STN
AN **93136902** MEDLINE
DN **93136902** PubMed ID: **1486392**
TI The treatment of Peyronie's disease with **tamoxifen**.
AU Ralph D J; Brooks M D; Bottazzo G F; Pryor J P
CS St Peter's Hospitals, London.
SO BRITISH JOURNAL OF UROLOGY, **(1992 Dec)** 70 (6) 648-51.
Journal code: 15740090R. ISSN: 0007-1331.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199302
ED Entered STN: 19930312
Last Updated on STN: 19930312
Entered Medline: 19930224

AB This is a preliminary study of the treatment of 36 patients with Peyronie's disease who received **tamoxifen** 20 mg twice daily for 3 months. An improvement occurred in 16 of 20 patients with penile pain, in 11 of 31 patients with an erectile deformity and 12 of 35 patients had a plaque shrinkage of at least 1 cm. Some improvement occurred in 6 of the 8 patients with a histologically confirmed inflammatory infiltrate of the plaque but not in any of the 4 patients without an infiltrate. The inflammatory infiltrate was found in patients in whom the duration of the disease was less than 4 months.

CT Check Tags: Human; Male
Adult
Aged
Middle Age
***Penile Induration: DT, drug therapy**
Penile Induration: PA, pathology
Penis: PA, pathology
***Tamoxifen: TU, therapeutic use**
Time Factors

RN **10540-29-1 (Tamoxifen)**

=> d all tot

L109 ANSWER 1 OF 2 MEDLINE on STN
AN 2000238189 MEDLINE
DN 20238189 PubMed ID: 10775130
TI Endothelium modulates anion channel-dependent aortic contractions to iodide.
AU **Lamb F S**; Barna T J
CS Department of Pediatrics, University of Iowa, Iowa City, Iowa 52242, USA.. fred-lamb@uiowa.edu
NC HL-62483 (NHLBI)
SO AMERICAN JOURNAL OF PHYSIOLOGY. HEART AND CIRCULATORY PHYSIOLOGY, (2000 May) 278 (5) H1527-36.
Journal code: 100901228. ISSN: 0363-6135.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200006
ED Entered STN: 20000706
Last Updated on STN: 20000706
Entered Medline: 20000628
AB Anion currents contribute to vascular smooth muscle (VSM) membrane potential. The substitution of extracellular chloride (Cl) with iodide (I) or bromide (Br) initially inhibited and then potentiated isometric contractile responses of rat aortic rings to norepinephrine. Anion substitution alone produced a small relaxation, which occurred despite a lack of active tone and minimal subsequent contraction of endothelium-intact rings (4.2 +/- 1.2% of the response to 90 mM KCl). Endothelium-denuded rings underwent a similar initial relaxation but then contracted vigorously (I > Br). Responses to 130 mM I (93.7 +/- 1.9% of 90 mM KCl) were inhibited by nifedipine (10(-6) M), niflumic acid (10(-5) M), **tamoxifen** (10(-5) M), DIDS (10(-4) M), and HCO(-)(3)-free buffer (HEPES 10 mM) but not by bumetanide (10(-5) M). Intact rings treated with N(omega)-nitro-L-arginine (10(-4) M) responded weakly to I (15.5 +/- 2.1% of 90 mM KCl), whereas hemoglobin (10(-5) M), indomethacin (10(-6) M), 17-octadecynoic acid (10(-5) M), and 1H-[1,2,4]oxadiazole[4,3-a]quinoxalin-1-one (10(-6) M) all failed to augment the response of intact rings to I. We hypothesize that VSM takes up I primarily via an anion exchanger. Subsequent I efflux through anion channels having a selectivity of I > Br > Cl produces depolarization. In endothelium-denuded or agonist-stimulated vessels, this current is

sufficient to activate voltage-dependent calcium channels and cause contraction. Neither nitric oxide nor prostaglandins are the primary endothelial modulator of these anion channels. If they are regulated by an endothelium-dependent hyperpolarizing factor it is not a cytochrome P-450 metabolite.

CT Check Tags: Animal; In Vitro; Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Aorta: DE, drug effects
***Aorta: ME, metabolism**
Bromides: PD, pharmacology
Calcium Channel Blockers: PD, pharmacology
Chloride Channels: AI, antagonists & inhibitors
Dose-Response Relationship, Drug
***Endothelium, Vascular: ME, metabolism**
Enzyme Inhibitors: PD, pharmacology
Ion Channels: DE, drug effects
*Ion Channels: ME, metabolism
Ion Transport: DE, drug effects
Nifedipine: PD, pharmacology
Norepinephrine: PD, pharmacology
Potassium Chloride: PD, pharmacology
Rats
Rats, Sprague-Dawley
Sodium Chloride: PD, pharmacology
Sodium Compounds: PD, pharmacology
*Sodium Iodide: PD, pharmacology
Sulfuric Acid Esters: PD, pharmacology
Vasoconstriction: DE, drug effects
***Vasoconstriction: PH, physiology**

RN 21829-25-4 (Nifedipine); 51-41-2 (Norepinephrine); 7447-40-7 (Potassium Chloride); 75-93-4 (methyl sulfate); 7647-14-5 (Sodium Chloride); 7647-15-6 (sodium bromide); 7681-82-5 (Sodium Iodide)

CN 0 (Bromides); 0 (Calcium Channel Blockers); 0 (Chloride Channels); 0 (Enzyme Inhibitors); 0 (Ion Channels); 0 (Sodium Compounds); 0 (Sulfuric Acid Esters)

L109 ANSWER 2 OF 2 MEDLINE on STN
AN 1998355830 MEDLINE
DN 98355830 PubMed ID: 9688908
TI Chloride ion currents contribute functionally to norepinephrine-induced vascular contraction.
AU **Lamb F S**; Barna T J
CS Department of Pediatrics, University of Iowa, Iowa City, Iowa, 52242, USA.
NC P30-HD-27748 (NICHD)
SO AMERICAN JOURNAL OF PHYSIOLOGY, **(1998 Jul)** 275 (1 Pt 2) H151-60.
Journal code: 0370511. ISSN: 0002-9513.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199808
ED Entered STN: 19980903
Last Updated on STN: 19980903
Entered Medline: 19980827
AB Norepinephrine (NE) increases Cl- efflux from vascular smooth muscle (VSM) cells. An increase in Cl- conductance produces membrane depolarization. We hypothesized that if Cl- currents are important for agonist-induced depolarization, then interfering with cellular Cl- handling should alter contractility. Isometric contraction of rat aortic rings was studied in a bicarbonate buffer. Substitution of extracellular Cl- with 130 mM methanesulfonate (MS; 8 mM Cl-) did not cause contraction. NE- and serotonin-induced contractions were potentiated in this low-Cl- buffer, whereas responses to K+, BAY K 8644, or NE in the absence of Ca2+ were

unaltered. Substitution of Cl- with I- or Br- suppressed responses to NE. Inhibition of Cl- transport with bumetanide (10(-5) M) or bicarbonate-free conditions (10 mM HEPES) inhibited NE- but not KCl-induced contraction. The Cl--channel blockers DIDS (10(-3) M), anthracene-9-carboxylic acid (10(-3) M), and niflumic acid (10(-5) M) all inhibited NE-induced contraction, whereas **tamoxifen** (10(-5) M) did not. Finally, disruption of sarcoplasmic reticular function with cyclopiazonic acid (10(-7) M) or ryanodine (10(-5) M) prevented the increase in the peak response to NE produced by low-Cl- buffer. We conclude that a Cl- current with a permeability sequence of I- > Br- > Cl- > MS is critical to agonist-induced contraction of VSM.

CT Check Tags: Animal; In Vitro; Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
4,4'-Diisothiocyanostilbene-2,2'-Disulfonic Acid: PD, pharmacology
Anions: ME, metabolism
Anthracenes: PD, pharmacology
Aorta, Thoracic: DE, drug effects
***Aorta, Thoracic: PH, physiology**
Bay-K-8644: PD, pharmacology
Bicarbonates: PD, pharmacology
Cell Membrane Permeability
Chloride Channels: AI, antagonists & inhibitors
*Chloride Channels: PH, physiology
*Chlorides: PD, pharmacology
Indoles: PD, pharmacology
Isometric Contraction: DE, drug effects
*Isometric Contraction: PH, physiology
Mesylates: PD, pharmacology
Muscle, Smooth, Vascular: DE, drug effects
***Muscle, Smooth, Vascular: PH, physiology**
Niflumic Acid: PD, pharmacology
*Norepinephrine: PD, pharmacology
Potassium: PD, pharmacology
Rats
Rats, Sprague-Dawley
Ryanodine: PD, pharmacology
Sarcoplasmic Reticulum: DE, drug effects
Sarcoplasmic Reticulum: PH, physiology
Serotonin: PD, pharmacology
Vasoconstriction: DE, drug effects
***Vasoconstriction: PH, physiology**
Vasodilator Agents: PD, pharmacology

RN 15662-33-6 (Ryanodine); 18172-33-3 (cyclopiazonic acid); 4394-00-7 (Niflumic Acid); 50-67-9 (Serotonin); 51-41-2 (Norepinephrine); 53005-05-3 (4,4'-Diisothiocyanostilbene-2,2'-Disulfonic Acid); 71145-03-4 (Bay-K-8644); 723-62-6 (9-anthroic acid); 7440-09-7 (Potassium); 75-75-2 (methanesulfonic acid)

CN 0 (Anions); 0 (Anthracenes); 0 (Bicarbonates); 0 (Chloride Channels); 0 (Chlorides); 0 (Indoles); 0 (Mesylates); 0 (Vasodilator Agents)

=> fil biosis
FILE 'BIOSIS' ENTERED AT 11:05:44 ON 27 AUG 2003
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FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 20 August 2003 (20030820/ED)

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=> d all

L117 ANSWER 1 OF 1  BIOSIS  COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN   1999:170544  BIOSIS
DN   PREV199900170544
TI   Tamoxifen normalizes the increase in vascular sensitivity
     associated with endothelial disruption.
AU   Liu, B.-X.; Barna, T. J.; Lamb, F. S.
CS   Univ. Iowa Pediatr., Iowa City, IA 52242 USA
SO   FASEB Journal, (March 12, 1999) Vol. 13, No. 4 PART 1, pp. A59.
     Meeting Info.: Annual Meeting of the Professional Research Scientists for
     Experimental Biology 99 Washington, D.C., USA April 17-21, 1999
     ISSN: 0892-6638.
DT   Conference
LA   English
CC   Pharmacology - Cardiovascular System  *22010
     Biophysics - Membrane Phenomena  *10508
     Metabolism - Minerals  *13010
     Cardiovascular System - Blood Vessel Pathology  *14508
     Pharmacology - Drug Metabolism; Metabolic Stimulators  *22003
     Pharmacology - Endocrine System  *22016
     General Biology - Symposia, Transactions and Proceedings of Conferences,
     Congresses, Review Annuals  *00520
     Biochemical Studies - Sterols and Steroids  *10067
     Biochemical Studies - Minerals  *10069
     External Effects - Physical and Mechanical Effects  *10612
     Endocrine System - Gonads and Placenta  *17006
     Laboratory Animals - General  *28002
     In Vitro Studies, Cellular and Subcellular  *32600
BC   Muridae  86375
IT   Major Concepts
        Cardiovascular System (Transport and Circulation); Pharmacology
IT   Parts, Structures, & Systems of Organisms
        aorta: circulatory system, endothelial disruption-induced sensitivity,
        tamoxifen normalization
IT   Chemicals & Biochemicals
          tamoxifen: cardiovascular - drug, chloride ion current
        inhibitor
IT   Miscellaneous Descriptors
        Meeting Abstract
ORGN Super Taxa
        Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
        rat (Muridae): animal model
ORGN Organism Superterms
        Animals; Chordates; Mammals; Nonhuman Mammals; Nonhuman Vertebrates;
        Rodents; Vertebrates
RN   10540-29-1 (TAMOXIFEN)
     16887-00-6 (CHLORIDE ION)


=> d his

     (FILE 'HOME' ENTERED AT 09:47:43 ON 27 AUG 2003)
               SET COST OFF

     FILE 'HCAPLUS' ENTERED AT 09:48:08 ON 27 AUG 2003
L1             2 S US20020065325/PN OR (WO2002-US26120# OR WO2000-US4892# OR US2
               SEL RN

     FILE 'REGISTRY' ENTERED AT 09:50:12 ON 27 AUG 2003
L2            17 S E1-E17
L3             1 S L2 AND C26H29NO
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L4          109 S C26H29NO/MF AND 46.150.18/RID AND 3/NR
L5           24 S L4 AND BUTEN? AND PHENOXY AND DIMETHYL AND ETHANAMINE
L6            5 S L5 NOT (LABELED OR (D OR T)/ELS OR 14C#)
L7            4 S L6 NOT 2 BUTENYL
L8            4 S L3,L7
                SEL RN
L9           24 S E18-E21/CRN
L10          14 S L9 NOT (COMPD OR WITH OR MXS/CI)
L11          18 S L8,L10
L12             STR
L13          18 S L12 CSS SAM
L14             SCR 2039 OR 2043 OR 2054
L15          16 S L12 NOT L14 CSS
L16         422 S L12 NOT L14 CSS FUL
                SAV L16 JKIM930/A
L17             STR L12
L18          11 S L17 CSS SAM SUB=L16
L19           4 S L16 AND C6-C6/ES
L20         235 S L17 CSS FUL SUB=L16
                SAV L20 JKIM930A/A
L21         182 S L20 AND 3/NR
L22          35 S L20 AND 4/NR
L23          18 S L20 NOT L11,L21,L22
L24           9 S L23 NOT (NC5/ES OR CLO4 OR MXS/CI OR C60H66N4O4 OR C20H8BR4O5

     FILE 'HCAPLUS' ENTERED AT 10:14:03 ON 27 AUG 2003
L25        5223 S L11
L26        7288 S TAMOXIFEN# OR ICI47699 OR ICI()(47699 OR 47 699)
L27        5795 S L19 OR L21 OR L22 OR L24
L28        7816 S L25-L27
                E BLOOD VESSEL/CT
L29       65303 S E3-E59
L30       13171 S E60-E96
L31        6190 S E109-E115
                E E3+ALL
L32      143069 S E5,E4+NT
                E E25+ALL
L33       92479 S E4,E5,E3+NT
                E E114+ALL
                E E28+ALL
L34        4260 S E3
                E E8+ALL
                E E30+ALL
L35        5383 S E3
                E E10+ALL
L36        9115 S E4
                E IMPOTENCE/CT
                E E3+ALL
L37        1385 S E2
                E ERECT/CT
                E E10+ALL
                E PENI/CT
                E PENIL/CT
                E E7+ALL
L38         446 S E2
                E PENIS/CT
L39        1507 S E3-E8
                E E3+ALL
L40        1902 S E6+NT
L41         324 S L28 AND L29-L40
L42         177 S L41 AND (PD<=19990226 OR PRD<=19990226 OR AD<=19990226)
L43         121 S L42 AND L25
L44         125 S L42 AND L27
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L45          125 S L43,L44
L46           52 S L42 NOT L45
L47            2 S L46 AND (NOREPINEPHRIN? OR CARDIOVASCULAR PATHOLOG?)/TI
                 E LAMB F/AU
L48            7 S E3,E11
L49           22 S E29,E31
                 E SCHUTTE B/AU
L50           57 S E3,E4,E7-E9
                 E YANG B/AU
L51          787 S E3-E24
                 E YANG BAO/AU
L52           18 S E3
L53           21 S E60
L54            4 S L28 AND L48-L53
L55           92 S L45 AND (PHARMACOL? OR PHARMACEUT?)/SC,SX
L56         2386 S (L11 OR L19 OR L21 OR L22 OR L24)(L)THU/RL
L57          551 S (L11 OR L19 OR L21 OR L22 OR L24)(L)(DMA OR PAC OR PKT)/RL
L58         2620 S (L11 OR L19 OR L21 OR L22 OR L24)(L)(BAC OR BCP OR BPR OR BSU
L59          104 S L45 AND L56-L58
L60          119 S L55,L59
L61            6 S L45 NOT L60
                 SEL L60 DN AN 3 8 9 27 74 81 93 119
L62            8 S L60 AND E1-E24
L63           11 S L1,L47,L54,L62 AND L25-L62
L64            4 S L63 AND (CL OR CL3 OR CLC3)(L)CHANNEL?
L65            3 S L63 AND (CHANNEL OR ION OR CHLORIDE OR CHLORIN?)(L)BLOCK?
L66            4 S L64,L65
L67            4 S L63 AND CHLORIDE(L)CHANNEL
L68            4 S L66,L67

     FILE 'REGISTRY' ENTERED AT 10:43:58 ON 27 AUG 2003
L69            1 S NOREPINEPHRINE/CN
L70            5 S C8H11NO3/MF AND NOREPINEPHRIN?
L71            3 S L70 NOT LABELED
L72            3 S L69,L71

     FILE 'HCAPLUS' ENTERED AT 10:45:21 ON 27 AUG 2003
L73           27 S L72 AND L28
L74           19 S NOREPINEPHRIN? AND L28
L75           10 S L73,L74 AND L42
                 SEL DN AN 3 5 8 9 10
L76            5 S L75 NOT E25-E39
L77            6 S L68,L76
L78            6 S L63 NOT L77
L79           12 S L77,L78 AND L1,L25-L68,L73-L78

     FILE 'REGISTRY' ENTERED AT 10:48:11 ON 27 AUG 2003

     FILE 'HCAPLUS' ENTERED AT 10:48:33 ON 27 AUG 2003
                 SEL HIT RN L79

     FILE 'REGISTRY' ENTERED AT 10:48:50 ON 27 AUG 2003
L80            3 S E40-E42

     FILE 'MEDLINE' ENTERED AT 10:49:16 ON 27 AUG 2003
L81         9336 S L11
L82        11681 S L26
L83         9336 S L27
L84         8664 S L81-L83 AND PY<=1999
                 E IMPOTENCE/CT
                 E E3+ALL
L85         8306 S E12+NT
                 E E11+ALL
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                E PENIL/CT
                E E44+ALL
L86          3462 S E5+NT
                E PENILE/CT
                E E8+ALL
L87          9608 S E4+NT
                E PENIS/CT
                E E3+ALL
L88         22633 S E5+NT
                E VASODILAT/CT
                E E5+ALL
L89         15145 S E5+NT
                E VASOCONSTRICT/CT
                E E4+ALL
L90         15236 S E5+NT
                E DIABETES/CT
                E E4+ALL
L91          4751 S E13+NT
                E DIABETES M/CT
                E E4+ALL
L92        160300 S E6+NT
                E HYPERTENSION/CT
                E E3+ALL
L93        159681 S E4+NT
                E BLOOD PRESSURE/CT
                E E3+ALL
L94        171519 S E5+NT
                E CORONARY ARTERY/CT
                E E28+ALL
                E E2+ALL
L95          8424 S E15+NT
                E E14+ALL
L96        131079 S E9+NT
                E VASCULAR/CT
                E E208+ALL
                E E7+ALL
L97        830845 S E3+NT
                E VASULATURE/CT
                E VASCULATURE/CT
                E VASCULAR TISSUE/CT
                E E4+ALL
                E ARTERIES/CT
                E E3+ALL
                E E3+ALL
L98        407473 S E3+NT
L99         59462 S E103+NT
L100           10 S L84 AND L85-L88
L101          249 S L84 AND L89-L99
L102            0 S L100 AND L101
                SEL DN AN L100 1-3 5
L103            4 S L100 AND E1-E12
L104           55 S L101 NOT AB/FA
L105          194 S L101 NOT L104
L106           98 S L105 NOT C4./CT

     FILE 'MEDLINE' ENTERED AT 11:02:24 ON 27 AUG 2003
L107            3 S L81-L83 AND (LAMB F? OR SCHUTTE B? OR YANG  B?)/AU
L108            2 S L107 NOT APOPTOSIS
L109            2 S L108 AND L81-L106

     FILE 'BIOSIS' ENTERED AT 11:03:44 ON 27 AUG 2003
                E LAMB F/AU
L110           58 S E3,E10,E12,E14
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                E SCHUTTE B/AU
L111        179 S E3,E4,E7-E10
                E YANG B/AU
L112        533 S E3-E25
                E YAND BAO/AU
                E YANG BAO/AU
L113         17 S E19
L114        766 S L110-L113
L115      11884 S L28
L116          3 S L114 AND L115
L117          1 S L116 AND CONFERENCE/DT

     FILE 'BIOSIS' ENTERED AT 11:05:44 ON 27 AUG 2003
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